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CHŌNĒ-CHONDROSTERNON

REPORT OF A CASE AND REVIEW OF THE LITERATURE

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CHŌNĒ-chondrosternon (funnel chest, pectus excavatum, koilosternia, trichterbrust, thorax en entonnoir) is derived from the three Greek words, *Χώνη Χονδρόστερνου*. This lesion is usually a congenital but rarely an acquired one. It consists of a depression of the lower portion of the sternum and the adjacent costal cartilages and characteristically becomes progressively more marked as the individual grows. The term chōnē-chondrosternon is considered preferable to trichterbrust,[1] torace imbubutiforme,[2] pecho infundibuliforme,[3] pecho en embudo,[4] pectus infundibuliforme,[4] thorax en entonnoir,[5] trechterbrost,[6] koilosternia,[7] and funnel chest, because it more accurately describes the lesion which consists of a funnel-like depression of the sternum and costal cartilages, whereas the others refer to a depression of the thorax without designating the position correctly. Pectus excavatum is an incorrect term, because pectus is the Latin word meaning breast and excavatum is the Latin word meaning hollow. The condition does not consist of a hollowing out of the breast, but consists of a depression of the lower portion of the sternum and the costal cartilages. The most descriptive term given to the condition is that by Snyder and Curtis[7] in which they use the term koilosternia, derived from two Greek words koilon and sternon. As the lesion consists of a funnel-shaped depression not only of the sternum, but also of the costal cartilages, it is our opinion that the term chōnē-chondrosternon is a more descriptive one.

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HISTORICAL

Bauhinus,[8] in 1594, described the condition of chōnē-chondrosternon and enumerated some of the clinical manifestations, such as dyspnea and paroxysmal cough. He described a case of a boy, seven years of age, in whom there was marked depression of the sternum and the ensiform cartilage. Johann Schenck,[9] who lived from 1531 to 1590, attempted to collect the literature on the subject.

Woillez,[10] in 1838, in his "Recherches" based upon measurements of the chest, described depressions of the sternum as "depressions sternaires," "la forme de la concavité d'un godet," "la depression en gouttière." It is impossible to determine from his description whether he refers to the typical condition of chōnē-chondrosternon or whether these were deformities of the chest produced by rickets.

Although the condition was recognized and described by Bauhinus[8] and Schenck,[9] relatively little attention was paid to this anomaly until 1860, at which time a number of communications appeared concerning the examination of a single patient. This case was a young medical student, Johann Heinrich Wojaczek, who was apparently matriculated in many European universities, including those of Vienna, Tübingen, Berlin, Breslau, and Paris. The first publication concerning this young gentleman was an anonymous one, which appeared in January, 1860.[11] In the same year Woillez[12] described this patient, who at that time was 22 years old. He stated that the patient had no symptoms and that no other members of the family had a similar condition. His deformity consisted of a depression of the lower end of the sternum which began in early childhood and at the age of 12 years began to increase in depth and extent. The patient was apparently unaware of the deformity until 1857 when, at the age of 19 years, Professor Rokitansky examined him and called his attention to the anomaly. Woillez's description at that time is still classic. He described the lesion as an enormous depression of the anterior portion of the thoracic wall, which is irregular, oval, and funnel-shaped. The depression measured about 25 cm. in a vertical direction, beginning at the manubrium sterni and the proximal ends of the clavicle and extending to the epigastrium. It measured 18 cm. in the transverse direction, a little below the nipples, which were located at the usual sites on the fourth rib and on the blunt edge of the depression. The walls of the depression were sharpest between the breasts and less definite toward the abdomen and in the subclavicular region. The bottom of the depression which was definitely funnel-shaped was 7 cm. deep and was located slightly to the left of the midline. From this depression three furrows extended, one to the manubrium, one below to the umbilicus, and another, which was horizontal, passed just beneath the left breast. Woillez[12] emphasized the fact that the patient had few symptoms. He thought that the condition was not congenital,

because the deformity increased with age and because he had never observed a similar depression in the examination of more than a thousand children. In 1863 Luschka[13] described the deformity occurring in Johann Heinrich Wojaczek, and his description is based on a plaster of Paris model made of the patient. According to Erich Ebstein,[14] this same patient also was described in a Viennese publication in 1867. Of interest, not only because of a similar deformity, but also because he presented himself at many of the German universities was a 24-year-old male, reported by Eggel[15] in 1870. Niemeyer,[16] in his presentation of this case, lamented the fact that there was no autopsy to prove his hypotheses concerning the pathogenesis of the condition. He stated that, whereas it should be differentiated from rachitic deformities, it develops as a result of an abnormal flexibility of the sternum which is probably caused by congenital nutritional disturbances or arrests in development. In 1872, Williams[17] described a case of a 17-year-old male who was born with the deformity and whose father and brother had similar conditions. In 1873, Flesch[18] reported a case of chōnē-chondrosternon which was observed in the Berlin outpatient clinic. Hagmann,[19] of Moscow, in 1880 also described the condition. In this same year, two publications emanated from Vienna, one by Langer and Zuckerhandl,[20] which was based on an anatomic investigation of the medical student Wojaczek, and an anonymous presentation[21] of this same individual. In the latter appeared a discussion by Zuckerhandl[21] and a description of the clinical findings by Breuner.[21] In 1882, Wilhelm Ebstein[1] collected five cases which had been reported and first gave the term "trichterbrust" (funnel chest) to the condition. This same author[22] reported another case of chōnē-chondrosternon in the same year. An excellent résumé of the subject was presented in 1909 by Erich Ebstein.[14]

Incidence.—True chōnē-chondrosternon occurs infrequently. Eichhorst[23] observed only six cases in seven years among 14,000 patients, an incidence of 0.04 per cent. Rosenfeld,[24] in ten years' time, observed three cases among 2,046 individuals whom he examined, an incidence of 0.14 per cent. Lombroso[25] found seven (1.06 per cent) cases among 659 prisoners whom he examined. According to Nowak,[26] of 30,000 Viennese school children there were twelve (0.14 per cent) with chōnē-chondrosternon. Thus, in a total of 46,705 persons there were twenty-eight cases with this deformity, an incidence of 0.059 per cent. This probably represents the true incidence of chōnē-chondrosternon. Lang,[27] in a recent presentation concerning the abnormalities of the thorax of children, states that of 3,488 boys examined, 2.2 per cent had this deformity. She also stated that in each instance the thoracic deformity was the result of rickets. It is questionable whether these cases should be considered as true chōnē-chondrosternon. It is our opinion that they represent rachitic deformities and are not true examples of this condition. Undoubtedly there has been a great deal

of confusion among clinicians concerning the characteristic manifestations of true chōnē-chondrosternon because of the other more frequent abnormalities of the thoracic cage. According to Sauerbruch,[28] Drachter,[29] in 1917, did more to designate clearly the condition of chōnē-chondrosternon than anyone else. Concerning the sex incidence, Eichhorst,[23] in thirty collected cases, found twenty-three (76.7 per cent) were males and seven (23.3 per cent) were females. Ebstein[14] collected from the literature ninety-seven cases of chōnē-chondrosternon, of which eighty-seven (89.67 per cent) were males and ten (10.37 per cent) were females. Of 268 collected cases in which the sex was stated, 220 were males and forty-eight were females, an incidence of 78.1 per cent and 21.8 per cent respectively.[7, 14, 26, 28, 30-55] It will thus be seen that the males predominate considerably, almost in the ratio of 4 to 1. Little can be said concerning the age incidence, because with the exception of a few isolated acquired cases, chōnē-chondrosternon is a congenital lesion and is present at birth or appears shortly thereafter.

Etiology.—Chōnē-chondrosternon is usually a congenital condition and is frequently inherited. In 1872, Williams[17] first called attention to the occurrence of this anomaly in several members of a family. The case which he reported was that of a 17-year-old boy who was born with the deformity and whose mother's father and one of his mother's brothers had the same anomaly. Since this time 118 other cases have been reported in thirty-one families. According to Peiper,[33] Troisier and Monnerot-Dumaine,[37] Nowak,[26] and Snyder and Curtis[7] this hereditary factor is dominant. Klemperer[56] reported five cases in a family in which the anomaly was present in four generations. Similarly, Nowak,[26] Troisier, and Monnerot-Dumaine[37] reported families in which the deformity was transmitted through four generations. In Nowak's series, in two families it was present in one generation, in seven families, in two generations, and in two families, in three generations. Of a total 121 members in twelve families reported by Peiper,[33] Troisier and Monnerot-Dumaine,[37] and Nowak[26] there were 49 (40.4 per cent) affected with this deformity. There can be no question about the anomaly being a dominant inherited characteristic in these families. In Nowak's[26] Case 12, four out of five in the family were affected, the father, two sons, and a daughter, the mother being the only unaffected member of the family. The cases reported by various authors showing hereditary tendencies are as follows: Vetlesen,[57] father and one of seven children; Kundmüller,[58] father and son; Smith,[59] mother and daughter; Herbst,[60] one family with two brothers, another family with two brothers and two sisters; Grünenthal,[61] two sisters and one brother and one girl cousin; Klemperer,[56] two brothers, mother, grandmother, and great-grandmother; Ramadier and Sérieux,[5] brother and sister; Anthony,[63] both of twin boys; Chlumsky,[64] two sis-

ters and one brother; Hoffa,[65] child and grandfather; Ebstein,[14] father and son; Groedel,[66] brother, two sisters, their father, his two brothers and grandfather; Taylor,[67] mother and daughter; Edlefsen,[68] four children of same family; Schümann,[69] four children, father and grandmother; Peiper,[33] mother, daughter and son (the daughter had one son affected, the son had one daughter and two sons affected); Troisier and Monnerot-Dumaine,[37] two brothers and a sister (one brother had a son, the sister had a son and a daughter, the son had two daughters and a son who had a grandson); Snyder and Curtis[7] stated that the condition occurred in eight members of the family they reported and was transmitted directly from parent to offspring. Nowak,[26] Case 1: Mother, daughter, son, and granddaughter; Case 2: father and son; Case 3: son, his mother, her father, his sister, and their father; Case 4: two brothers; Case 5: father, son, and daughter (the son had a daughter and the daughter had two sons); Case 6: father, son, and daughter; Case 8: brother and sister; Case 9: grandmother and granddaughter; Case 10: brother and sister; Case 12: father, two sons, and a daughter. Curschmann[53] reported father, son, and daughter. From this high incidence of reported cases in which chōnē-chondrosternon is definitely a familial trait, it becomes obvious that at least in some cases heredity plays a conspicuous etiologic role. However, attention must be directed to the fact that there have been a large number of cases observed in which exhaustive inquiries of the families revealed no other member affected with this unsightly anomaly. The authors' case is an example of this latter group.

Growth disturbances are thought by many to be the cause of chōnē-chondrosternon. Ebstein,[1] in 1882, expressed the belief that the condition was due to an arrest in development of the sternum, the sternum growing less rapidly and, therefore, being of smaller size and shorter length than would normally occur. He based this hypothesis on the findings in a case reported by Hagmann,[19] in which a patient, 9 years of age, had a body length 131 cm. whereas the sternal length was 7.4 cm. Ebstein states that this length of 7.4 cm. is equivalent to a 1-week-old baby according to the anatomic investigations of Wintrich. Versé[70] also expressed the belief that an abnormal growth in length of the body of the sternum caused the deformity. On the other hand, Erich Ebstein[14] refutes this theory because he states in agreement with Strauch[71] that it is extremely difficult in life to measure accurately the sternal length. In complete accord with Matterstock,[72] he concludes that there is no normal relationship between the length of the sternum and the body length. According to Matterstock's[72] investigation on 100 normal men ranging from 15 to 30 years of age, the length of the sternum, including the xiphoid process, varies from 15.5 to 24 cm. Niemeyer,[16] in discussing the case presented by Eggel,[15] is of the opinion that the deformity is due to some nutritional disturbance or to a developmental retardation, which

results in an abnormally flexible sternum. As the sternum is fixed in the upper portion by the ribs and clavicles, the atmospheric pressure exerting its effect on the lower, less fixed flexible portion results in a depression. Féré and Schmid[73] also believed that there occurred some disturbance in the nutrition of the bone, causing an arrest in development and consequent increase in flexibility thus diminishing its ability to withstand atmospheric pressure. Müller,[74] in describing the embryologic development of the thorax, showed that in the 32 mm. embryo there is a depression of the sternum beginning at the third chondrosternal junction and becoming progressively deeper to the seventh. In the 37 mm. embryo, this normal deformity is increased. Similarly Bien[75] observed that such depressions in the chondrosternal region occur normally in the development of the embryo and stated that this physiologic trichterbrust may persist until birth and actually increase thereafter. According to Flesch,[18] who presents a typical case, this deformity is due to a disturbance in the physiologic growth of the thorax. He quotes Schiffer, his chief, as believing that it is due to an overgrowth of the ribs which in turn results in a depression of the sternum. Marie[76] is also of the opinion that the lesion is due to a disturbance in growth resulting in overgrowth of the ribs and depression of the sternum. Closely related to these theories of Niemeyer,[16] Eggel,[15] Féré and Schmid[73] is that of Picqué and Colombani,[77] who believe that the deformity is due to a persistence of an abnormal flexibility of the sternum. They account for the persistence of the flexibility on the basis of the disturbance in the ossification of the lower portion of the body. Normally, ossification occurs in the manubrium in the eighth month of fetal life, whereas that in the lower portion of the sternum does not occur until after the tenth month after birth. Nageotte-Wilbouchewitch[78] also believes that the lesion is due to a congenital aplasia of the sternum. She states that in the normal newborn infant who might have an inspiratory obstruction the osteocartilaginous portion of the thoracic cage does not become deformed. If, however, the infant is premature or debilitated, even in the absence of the obstruction, because of the congenital aplasia and resultant greater flexibility of the sternum, there is retraction of the lower portion of this part of the thorax. Hagmann[19] is of the opinion that the condition may be the result of an abnormal loosening of the chondrosternal articulation.

Curschmann[53] recently reported a case of chōnē-chondrosternon occurring in a patient with arachnodactylia, which, according to Brissaud,[79] is due to a pituitary disturbance occurring in the fetus, the same disturbance producing in the adult, acromegalia, and in the child, gigantism. The patient, reported by Curschmann,[53] was a 23-year-old male with marked arachnodactylia ectopia of the lens and chōnē-ćhondrosternon, whose father and sister had a similar thoracic defect, the sister having also arachnodactylia. This case is of particular in-

terest to us, because our case, which is included in this report, also had an arachnodactylia without lens ectopia. In this regard, Bauer[80] stated that chōnē-chondrosternon is a constitutional anomaly and is probably due to degenerative diseases.

According to Woillez,[12] the most likely cause of chōnē-chondrosternon is retraction of the central diaphragmatic tendon. He compares this lesion with Dupuytren's contracture of the fingers. Spitzy[81] and Hofbauer[82] also designate this mechanism as an explanation for this developmental anomaly. In Hutchinson's opinion[83] the deformity is the result of fixation of the lower end of the sternum by fibrous bands to the central tendon of the diaphragm during the development of the thorax, the remainder of the thorax developing normally and assuming its normal shape while the lower portion of the sternum remains fixed. A similar mechanism is that which occurred in the case reported by Habs[84] in which a child, 5 months old, had a congenital shortening of the rectus abdominus with apparently a typical chōnē-chondrosternon. Following sectioning of the muscle immediately after operation the deformity of the chest was relieved. Habs[84] is of the opinion that the shortening of the rectus abdominus was due either to nerve stimulation or to an inflammatory process. Depression of the sternum is thought to be due in some cases to retrosternal adhesions as a result of a mediastinitis. Raubitschek[85] believes that one of his cases was the result of a mediastinitis although he was unable to prove it. Jacobi[86] was of the opinion that the mediastinitis originated in the mediastinal lymph nodes, resulting in adhesions which in turn caused depressions of the sternum due to contraction of these fibrous bands. Richter[87] observed a young man, who as a child had a pericarditis and who subsequently developed a chōnē-chondrosternon, which Richter believed was due to adhesions in the anterior mediastinum. On the other hand Ribbert,[88] Marchand,[89] Ramadier and Sérieux[5] believe that mediastinitis has nothing to do with the development of this deformity because in their autopsy cases they were unable to demonstrate adhesions. In the authors' case, which was operatively relieved, the adhesions between the posterior surface of the sternum and the costal cartilages and the anterior mediastinum were exceedingly dense, in fact so dense that the sternum and costal cartilages were freely mobilized only with considerable difficulty. In connection with the mediastinal adhesions it is of interest that Chapard[90] reported a case of chōnē-chondrosternon developing following a mediastinal tumor in a child.

Numerous observers have suggested that pressure on the flexible thorax of the developing fetus while still in the embryo is responsible for the deformity. Langer and Zuckerhandl[20] and Kaumheimer[91] believed that the deformity was due to depression of the sternum by the chin, resulting from flexion of the neck. Hagmann[19] thought that the deformity in one of his cases was due to pressure of the heel of the

child on the sternum and costal cartilage. Marchand[89] reports a case in which the knee of the child fit the depression exactly. He is of the opinion that this is responsible for the abnormality. Erich Ebstein[14] reported a case in which the deformity was apparently caused by pressure of the flexed knee. Raubitschek[85] reported the occurrence of this anomaly in one of two newly born twins and believed that pressure of the head of one fetus on the anterior thorax of the other resulted in the deformity. This, however, was only temporary, as the condition disappeared within two weeks. From examination of the drawing and illustrations of Gerstenberg,[92] Erich Ebstein expressed the belief that the deformity in his case was due to pressure on the anterior thorax by an arm and elbow. On the other hand, W. Ebstein[22] is of the opinion that intrauterine pressure cannot be responsible for these deformities, because he maintains if the pressure were great enough to produce deformity of the chest, there would occur first compression of the cord and death of the fetus. Ribbert[88] was able to demonstrate in a 5-day-old child on whom he performed an autopsy that the depression of the thorax corresponded exactly to the contour of the lower jaw. The skull in this child showed considerable deformity which was a result of compression of the bones, which Ribbert stated was not due to pressure during labor but was the result of a long-continued influence of the growth of the skull in utero. Bien,[75] as the result of her anatomic investigations, concludes that pressure of the chin on the sternum cannot be responsible because she states that at the time the sternum develops, the chin of the fetus whose head is markedly flexed is in no position to produce the deformity of the lower end of the sternum. Appert[93] also suggested that intrauterine compression of the developing embryo may produce this structural defect. According to Graeffner[94] the abnormality is the result of trauma occurring during the latter months of pregnancy which may be a single severe injury or repeated shaking-up of the mother. Erich Ebstein[14] states that the hypothesis that continued trauma to the fetus in utero may produce these defects is substantiated by the fact that frequently other congenital anomalies, such as dislocation of the hip, are occasionally associated with the thoracic deformities. Such cases have been reported by Chlumsky,[64] Goesche,[95] and Marchand.[89] Deformities of the extremities have been reported by Ramadier and Sérieux,[5] W. Ebstein,[1] Grünenthal,[61] and Ebstein.[14] Flusser[43] believes that this deformity may be the result of injury to the laryngeal nerves during delivery, which in turn produces difficulty in inspiration and consequent increase in the negative pressure within the thorax thus causing the sternal retraction during the early formative stage.

Hereditary syphilis has been considered a causative factor in the development of chōnē-chondrosternon. Legrain[96] was the first to call attention to this possibility. He reported two cases with this deformity having definite evidence of congenital syphilis. In two additional

cases there were also signs of congenital syphilis. Chlumsky[64] reported three cases from Hoffa's private practice, all of whom were offsprings of a syphilitic mother. They were all more or less degenerates; one had hemoplegia with a normal mentality, the other was anemic and scoliotic. Gaucher,[97] Hallapeau and Tesseire,[98] Fournier,[98] Milan,[100] and Crouzon[101] all believe that congenital syphilis plays an etiologic role in the development of chōnē-chondrosternon.

In addition to the congenital causes described above, there are some cases which are apparently acquired. Of various acquired causes, rickets is by far the most important, although there is considerable controversy concerning the role which rickets plays. Many of the cases of thoracic deformity resulting from rickets are not true cases of chōnē-chondrosternon and should not be classified as such. Ribbert,[88] in his description in 1884, maintained that the condition had nothing to do with rickets. On the other hand Grünenthal,[61] Picqué and Colombani,[77] Hutchinson,[83] Clément,[102] Maunescie,[103] Steindler,[104] Bystrow,[105] and Noica[106] believe that rickets plays a prominent role in the development of this deformity. Lang[27] examined 3,488 school boys, 2.2 per cent of whom had what he called funnel chest. He also examined 3,145 school girls, 2.5 per cent of whom had the same anomaly. In each instance there was evidence of rickets. It is very questionable, as mentioned above, whether these anomalies can be considered as true chōnē-chondrosternon, or deformities produced by rickets.

In addition to rickets, other acquired causes of chōnē-chondrosternon are traumas. These traumas may consist of a single injury resulting in depression of the anterior thorax which remains permanent and progressive or which may be the result of repeated traumas which occur primarily in occupations. Seupel[107] reports a case in which the deformity was presumably caused by pressing of the thumb on the infant by the midwife during resuscitation, resulting in depression of the anterior wall of the thorax, which persisted and became augmented. Two cases of traumatic chōnē-chondrosternon which were treated operatively were reported by John Alexander.[38] MacAusland and Tighe[108] report a case of depressed fracture of the sternum in its mid-portion which was reduced by traction five days later resulting in complete relief of symptoms. It is our belief that this could not be termed a case of chōnē-chondrosternon, but should more accurately be termed a fracture of the sternum. In those cases, however, due to lack of reduction in which the deformity persists, one might be justified in considering it a funnel chest. Bernhardt[109] reported a case of "trichterbrust" in a man 35 years of age, whose deformity was caused by a fall down some steps at the age of 6 years. A somewhat similar case was reported by Wolostnick[110] in a boy 6 years of age whose abnormality was caused by a fall at the age of 2 years. As early as 1819, Laennec[111] called attention to the deformity of the chest

resulting from pressure, particularly in those occupations in which pressure is likely to occur, as in cobblers, potters, and tailors.[112] Eichhorst[23] reports a case occurring in a railroad worker, who apparently developed a deformity of the chest as a result of pressure on the anterior wall of the thorax by the car bumpers.

According to Kuhns,[113] faulty posture may be a precipitating factor in chōnē-chondrosternon. In contradistinction to the normal individual with correct posture in whom during inspiration there occurs an elevation of the ribs and the sternum, children of slender skeletal build and faulty posture manifested by a slumping forward of the shoulders and a lateral position of the scapulas lose the elevating action of the pectoralis muscles on the sternum. The forward bowing of the dorsal spine causes a flattening of the chest, and the lowering of the diaphragm results in serious mechanical difficulties of respiration. In such a position the central tendon occupies a lower position, resulting in a pull on the lower end of the sternum during inspiration. Conforming with Kuhns' conception of postural defects are the conceptions of those who believe that interference with respiration due to other causes might be responsible. Redard,[114] in 1890, reported a case of chōnē-chondrosternon in a child 13 years of age with gradual improvement after removal of hypertrophied tonsils and adenoids. Similar cases have been reported by Chapard,[90] Kirmisson,[115] and Karewski.[116] Pironneau[117] stated that hypertrophy of the pharyngeal lymphatic ring may be the causative factor. Codman,[118] in 1901, also suggested that the deformity might be the result of respiratory obstruction produced by adenoids or asthma. Others who have made such suggestions are van den Bergh[119] and Osler and McCrae.[120]

Cases have been reported in which the deformity was presumably the result of developmental defects in the neuromuscular system. Landouzy and Dejerine[121] reported a case, in 1885, which was associated with progressive muscular atrophy. A similar case was reported by Schultze.[122] Guinon and Souques[123] observed that four of five patients presenting this deformity had progressive myopathy. Frühwald,[124] on the basis of two cases presenting these abnormalities which he observed, suggested that anomalous development of the pectoral and rectimuscles may play an etiologic role.

Pathology.—Chōnē-chondrosternon is essentially a structural abnormality of the thoracic cage with resultant distortion and displacement of the thoracic and upper abdominal viscera. The skeletal deformity consists of a funnel- or oval-shaped depression of the body of the sternum and its adjacent costal cartilages. As emphasized by Chlumsky,[64] the depression is circular in the advanced cases and oval in the less accentuated ones. The depression usually begins above at the junction of the manubrium with the body of the sternum, and becomes progressively deeper as one passes caudad to about the level of the tenth dorsal vertebra where the greatest depth exists. The

deepest part of the depression according to most authors is slightly caudad to a line drawn between the two nipples which is approximately at or slightly above the sterno-xyphoid articulation. The deformity consists not only of depression of the sternum and costal cartilages but also of an axis deviation, either to the right or left, of the former. When such does occur, the anterior surface of the sternum faces the smaller half of the thorax (Fig. 1). The deviation of the sternum is usually such that the anterior surface faces the right side. Because of the chondrosternal depression an angulation between the costal cartilages and ribs is produced, this angulation forming the rim of the funnel laterally. The increased chondrosternal angulations result in decreased mobility of the thorax because the normal elevation of the ribs during inspiration is interfered with. This interference with respiration is thought by many to predispose to chronic respiratory tract infections such as tuberculosis. Because of the axis deviation of the sternum, the costochondral angulation varies on the two sides, being more acute on the side which the anterior surface of the sternum faces and more obtuse on the opposite side. On the former

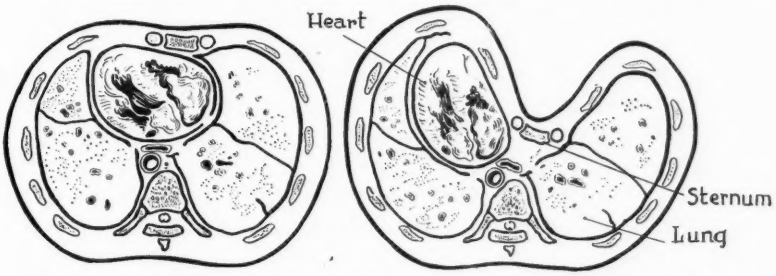


Fig. 1.—Diagrammatic illustration of cross section of chest at level of fourth costal cartilage in the normal (left) and in authors' case (right) as based upon cyrtometric measurements. The left hemithorax is larger than the right. There is a deviation of the sternum to the right on its longitudinal axis, an increase in the anteroposterior diameter of each hemithorax in the mamillary line, and an increase in the transverse diameter of the thorax. The heart is displaced to the left and is entirely in the left hemithorax.

the wall of the funnel is steeper, whereas on the latter it is more sloping. As emphasized by Woillez[12] the lateral walls are steepest, whereas those portions of the funnel-shaped depression which are located cephalad or caudad have more gradual slopes. The depth of the depression varies considerably but usually measures from 7 cm. to 10 cm. According to Picqué and Colombani[77] a depression of less than 7 cm. depth should not be considered as a true example of chōnē-chondrosternon. They suggest the capacity of the depression in recording cubic centimeters of water. They found that this varies from 50 c.c. to 170 c.c. or more.

We agree with these authors that chondrosternal depressions of slight degree should not be considered chōnē-chondrosternon; but if one were to adhere to this measurement most of the reported cases

would be excluded. Obviously in considering measurements of the depression one must take into consideration the age of the patient. The length and breadth also vary considerably. In length it varies from a few centimeters to 20 cm. or more and in width it varies from a few centimeters to 12 cm. In the authors' case, the length of the depression was approximately 18 cm., the width was approximately 11 cm., and the depth was about 7.5 cm. Because of the chondrosternal depression, the sterno-vertebral thoracic diameter becomes markedly decreased. This distance varies considerably depending upon the age of the individual and the degree of depression. In Versé's[70] patient, who was an adult female, the distance between the posterior surface of the sternum and the anterior surface of the vertebral body at the greatest depth of the depression was only 1.5 cm. In the authors' case it was estimated at the time of the operative correction of the deformity that this distance was approximately 2 cm. The encroachment upon the thoracic cavity in the sterno-vertebral diameter is compensated for, as first emphasized by Eggel,[15] in the following manner: (1) increase in the anterior posterior diameter of each hemithorax in the mammillary line; (2) increase in the transverse diameter of the thorax; (3) increase in the vertical diameter (length) of the thorax produced by a more caudal position of the diaphragm (Fig. 1).

As the result of the decrease in the sterno-vertebral diameter, displacement and distortion of the mediastinal structures and other viscera occur. The heart is displaced to the left and usually lies entirely within the left hemithorax. In addition to its displacement the heart is also rotated around its longitudinal axis as demonstrated by Groedel[66] and Stadtmüller.[36] In addition to torsion produced by rotation of the heart in its long axis, lateral rotation can occur resulting in kinking of the large vessels at the base of the heart. Cases with such lesions have been reported by Versé[70] and Ombrédanne.[40] The cardiac displacement was so great in Ebstein's,[22] Rosenfeld's[24] and Edeiken and Wolferth's[42] cases that the apex beat was in the mid-axillary line. Henschen and Naegeli[125] observed a case in which a depression of the right ventricle was caused by the thoracic deformity. Bien[75] observed a case in which the base of the heart was so compressed by the funnel-shaped deformity that a depression was present in the right auricle extending to the right ventricle. The length of the auricle was also increased by the compression.

Occasionally, as emphasized by Pohl,[126] in spite of the diminished sterno-vertebral distance the heart is not displaced but retains its retrosternal position because of fixation by mediastinal adhesions. Edeiken and Wolferth[42] also observed such a case in which the heart was not displaced. As the patient had had an antecedent pericarditis, they assumed the resultant adhesions prevented the cardiac displace-

ment. Still more rarely, in those cases in which the chōnē-chondrosternon is predominantly on the left, the heart may be displaced to the right as occurred in Edeiken and Wolferth's Case 9.[42]

The mediastinal interpleural space becomes increased in width as the result of the depressed anterior thoracic wall, causing a transverse compression of the lungs on both sides. Because of the more caudad position of the diaphragm, the vertical diameter of the lungs becomes increased. Probably as the result of the decreased sternovertebral diameter, pressure is exerted upon the relatively fixed mediastinal structures. Because of compression of the esophagus, dysphagia occurs. Ombrédanne[40] observed a case with partial obstruction of the esophagus. In the authors' case, the inferior vena cava and aorta were displaced to the right and left sides, respectively. Because of the pulmonary compression caused by the deformity and the rigidity of the thorax resulting in diminished mobility, mentioned previously, chōnē-chondrosternon is frequently accompanied by decrease in the vital capacity. Generally this is not marked. In one of Rocher and Laporte's cases,[39] however, the vital capacity was only 1,000 c.c., and in a case reported by Fabre[127] it was only 900 c.c. According to Kuhns[113] the vital capacity is often half the normal value.

In addition to the displacement and distortion of the thoracic viscera, similar disturbances may exist in the upper abdominal viscera. The liver is usually displaced downward and, as shown by Stadtmüller's case,[36] occasionally is compressed. Alterations in the configuration of the vertebral column are frequently associated with chōnē-chondrosternon. These consist of an exaggeration of the normal dorsal kyphotic and lumbar lordotic curves and lateral deviation. The lordosis is determined by the longitudinal rotation of the sternum, the concavity being toward the side which the anterior surface of the sternum faces.

Clinical Manifestations.—Most of the reported cases of chōnē-chondrosternon have had few or no symptoms. Usually only in those cases in which the depression is marked are symptoms present. When present, symptoms are the result of displacement, distortion, or compression of the thoracic or upper abdominal viscera. The symptoms can be classified into four groups depending upon the visceral involvement. The most important clinical manifestations aside from the deformity are those referable to the cardiovascular system, of which dyspnea, palpitation, and precordial pain are the most conspicuous. Sauerbruch[28] reported a case of paroxysmal fibrillation which was relieved by operative correction of the deformity. In cases in which the deformity is allowed to continue, even cardiac failure can occur. On the other hand, Groedel[66] believes that the cardiac failure occurring in cases of chōnē-chondrosternon is not the result of the thoracic deformity. The potential danger of this anomaly is well illustrated by

one of Chlumsky's patients[64] whose life was saved by the prompt removal of a left-sided pleural exudate which produced threatened cardiovascular failure because the heart could not be displaced to the right side. A somewhat similar case was reported by Béclère.[128]

Soft systolic murmurs which are not transmitted and are usually functional are not infrequently heard over the mitral area. Electrocardiographic findings described by Edeiken and Wolferth[42] were normal in seven of their ten cases. In two cases there was evidence of depressed myocardial function and in one there was right axis deviation which they interpreted as being due to a primarily left-sided depression. Rocher and Laporte's case[39] of chōnē-chondrosternon showed no electrocardiographic evidence of heart disease. Edema is an infrequent finding in this anomaly. Edeiken and Wolferth's[42] Case 6 had edema of the lower extremities which undoubtedly was due to cardiovascular involvement.

As the result of pulmonary compression, respiratory symptoms can occur. These consist of dyspnea (which can also be cardiac), cyanosis, and cough. The case reported by Gerstenberg[92] was a patient with cyanosis of the lips. As mentioned previously there may be diminished vital capacity, in some cases quite considerable diminution. Respiratory infections are likely to occur in these patients, probably because of the decreased pulmonary ventilation. In addition to cardiorespiratory manifestations chōnē-chondrosternon may be accompanied by digestive disturbances. Dysphagia, because of esophageal compression, is the most frequently encountered symptom. Ombrédanne[40] reports a case in which the thoracic depression resulted in partial esophageal obstruction.

Dyspnea is a rare complaint. Its occurrence can be explained by gastric displacement, and also possibly because of dysfunction resulting from vagal compression within the mediastinum. The general physical status of patients with this impairment is usually fairly characteristic. They have an asthenic habitus, are pale and hyposthenic. According to Kuhns[113] these factors are predisposing to chōnē-chondrosternon and are not the result of the condition. It is difficult to determine which is primary in those patients characterized by these features, but it cannot be denied that this deformity can and does occur in individuals who are sthenic, robust, and otherwise perfectly well. Curschmann[53] has called attention to the possible association of this anomaly with arachnodactylia and describes a family in which the father, a son, and daughter exhibited this association. The authors' case is also a typical example of arachnodactylia with chōnē-chondrosternon (Fig. 2). It is of interest that although no other members of the patient's family have the thoracic deformity, her father and his family, her two brothers, and three sisters are very tall and slender.

Differential Diagnosis.—The diagnosis in a typical case of chōnē-chondrosternon is so obvious that no difficulty should be encountered in establishing its identity. Other deformities of the thorax have been confused with the classical form of this anomaly. These include particularly rachitic deformities and congenital absence of the pectoral muscles and ribs.

Whereas sternal depression resulting from trauma may simulate clinically true chōnē-chondrosternon, it should be considered as a separate entity.

The classical form of chōnē-chondrosternon consists of a congenital funnel-shaped depression of the lower end of the sternum and the adjacent costal cartilages which usually becomes progressively worse as the individual grows.

Fig. 2. Fig. 3.

Fig. 2.—Photograph of authors' patient made following operation. Patient displays characteristics of arachnodactylia.

Fig. 3.—Roentgenogram of chest of authors' patient in the lateral view before operation. The marked depression of the sternum is outlined. There is a slight increase in the normal dorsal kyphotic curve.

Roentgenologic examination usually reveals the characteristic features of the anomaly. In the anteroposterior view the slight lateral curvature of the vertebral column can frequently be demonstrated. The heart shadow usually shows displacement to the left and may appear somewhat larger than normal. Rösler[129] has directed attention

to the reduction in ratio of the anteroposterior to the transverse diameter of the heart from the normal of 0.7 to 1 to 0.5 to 1 or less. Both leaves of the diaphragm may have a considerably lower position than normal. In the lateral view the marked depression of the sternum is clearly visible as well as the exaggerated normal dorsal kyphotic curve (Fig. 3).

Complications.—Complications of chōnē-chondrosternon may be classified into two groups depending upon the viscera involved.

Cardiovascular complications consist of myocarditis and cardiac hypertrophy. Appert[93] described a case with a probable pulmonary stenosis and Roulucroix and Manel[130] reported one with mitral stenosis. Larini[131] reported evidence of concomitant congenital cardiac disease and chōnē-chondrosternon.

Obviously it cannot be determined whether these cardiac lesions represent complications of the thoracic anomaly or whether they occurred coincidentally.

Respiratory tract infections have been reported frequently as complications of chōnē-chondrosternon. These are thought to be the result of diminished pulmonary aeration because of the compression of the lungs and the rigidity of the chest wall. Sato[132] believes that alterations in the shape and movement of the thorax bear a direct relationship to the development of tuberculosis. This probably accounts for the development of tuberculosis in chōnē-chondrosternon. Dittrich[133] accounts for the tendency toward bronchial inflammatory disturbances in these patients by their shallow breathing. Erich Ebstein,[112] Meyer,[30] Mendez,[3] Hoffmeister[35] and Shively[31] recorded the occurrence of pulmonary tuberculosis in patients with this anomaly. Shively found tuberculosis in ten of seventeen patients with chōnē-chondrosternon. Chronic bronchitis and asthma are not infrequently observed in this condition.

Treatment.—The very mild forms of chōnē-chondrosternon which produce no subjective or objective disturbances require no therapy. However, in the more severe types causing cardio-respiratory embarrassment and subjective complaints and disability, some form of corrective procedure is indicated. The various methods of therapy which have been devised for this unsightly and frequently incapacitating abnormality may be classified into two groups: (1) conservative and (2) operative.

Until 1911, when Ludwig Meyer[30] first performed an operative procedure in an attempt to relieve a patient with chōnē-chondrosternon, the treatment of this condition consisted only of conservative procedures, i.e., breathing and postural exercises and orthopedic measures. In 1903, Richard[134] reported two cases, one of which had an associated incipient pulmonary tuberculosis with decided improvement following breathing exercises. Cotton[135] also recorded two cases

relieved by breathing exercises. Ober[136] states that the condition responds very well to breathing exercises and to the placement of the patient on a hyperextended frame. Möhring[137] and Steindler[104] also advocate thoracic exercises. Taylor[67] suggests thoracic exercises devised to increase the strength of the posterior muscles of the back and shoulder girdle. Kuhns,[113] on the basis of his conception of the etiology, advises avoidance of excessive fatigue, remedying of faulty posture, and maintenance of good carriage. Roederer[138] also maintains that considerable improvement can be obtained by the use of postural exercises and thoracic appliances. Adhesive traction on the depressed sternum and the application of suction to the anterior thoracic wall in an attempt to correct the deformity have been utilized by Hoffa[65] and Clément[102] with indifferent success. According to Glaeser[139] purely conservative measures should be considered only for young patients with yielding bones. Conservative procedures should always be tried, and undoubtedly in many cases, particularly in the milder forms, considerable improvement can be obtained.

The institution of operative therapy in chōnē-chondrosternon is justified only in those severe cases in which definite manifestations of cardiorespiratory disturbances are present. The fact that only thirty-two patients have been operated upon in the past twenty-seven years clearly reveals how infrequently surgical intervention is indicated. However, this low incidence of operative cases does not necessarily indicate that the incidence of cases requiring operation is commensurably low. Undoubtedly there have been a greater number of patients requiring operation who for varying reasons never received the benefit of surgical relief. To a certain extent this has been due undeniably to a recalcitrant attitude on the part of physicians as well as patients to accept operative measures and even to failure of appreciation of the relief that may be obtained by surgical procedures. This latter statement is exemplified by the publication of Fischer,[140] who wrote, in 1928, that "up to the present time the measures employed do not appear to have produced any appreciable results; it is therefore a structural defect which must be conserved."

In 1911 Ludwig Meyer[30] performed the first operation for chōnē-chondrosternon. The patient, a 16-year-old boy, complained of marked dyspnea and had an associated pulmonary tuberculosis. Meyer, after consulting Freund, resected about 2.5 cm. of the second and third right costal cartilages (Fig. 4). Although the patient was relieved of the dyspnea on the second postoperative day, the end result was not satisfactory. As the symptoms of this anomaly are due to displacement, distortion, or compression of the thoracic viscera by the depressed sternum and adjacent costal cartilages, the operative relief of the condition obviously must consist of either elevation (correction of the de-

pression) or the removal of the involved chondrosternum. The explanation for the unsatisfactory result obtained by Meyer, therefore, is readily apparent.

The next attempt to relieve chōnē-chondrosternon by surgical intervention was made by Sauerbruch[28, 41] in 1913. The patient, an 18-year-old boy, had a congenital "trichterbrust" and complained of marked dyspnea and palpitation. The operation which Sauerbruch performed consisted of resection of the fifth to the ninth costal cartilages on the right side, a portion of the corresponding ribs, and part of the adjacent sternum (Fig. 5). Sauerbruch states that a satisfactory result was obtained in this case as well as in three subsequent cases in which a similar operation was performed.

In 1925, Zahradnicek[34] reported a case of marked chōnē-chondrosternon in a boy, 16 years of age, complaining of dyspnea and palpitation, with a satisfactory result following operation. The procedure he performed consisted of introduction of two wires through perforations in the sternum and the application of traction (Fig. 6). After the operative experience in the case reported the authors agree with Alexander[38] and Garnier[141] that it is difficult to understand how mobilization of the unyielding depressed chondrosternum could be obtained satisfactorily by such a simple procedure.

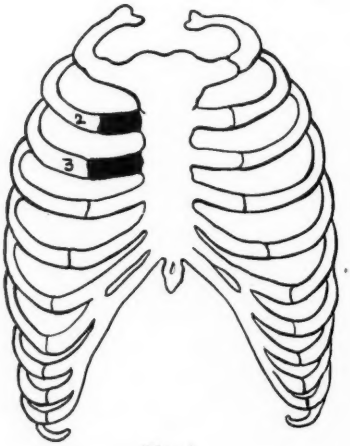


Fig. 4.

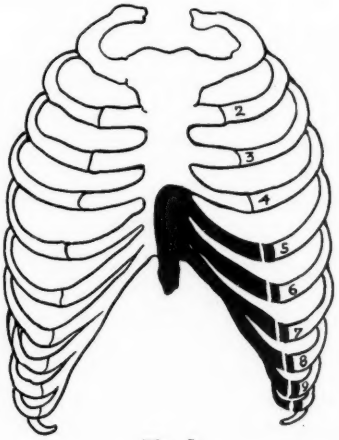


Fig. 5.

Fig. 4.—Diagram illustrating operation performed by Meyer,[30] 1911, for chōnē-chondrosternon. Black area represents costal cartilages which were resected. (One case, unsatisfactory.)

Fig. 5.—Diagram illustrating first operation performed by Sauerbruch,[28] 1913. Black area represents parts resected. (Four cases, successful.)

Hoffmeister, in 1927, reported a successful operative result in a 19-year-old male patient operated upon by Lexer. The operation consisted of resection of the fifth to the ninth costal cartilages on both sides and section of the sternum at the level of the fifth chondrosternal junction

(Fig. 7). The sternum was then turned around so that the anterior surface became posterior in order to protect the heart. In 1931, Alexander reported two cases of "traumatic pectus excavatum" one of which was in a boy, 16 years old, who was injured four years previously while wrestling. The operation in this case consisted of a T-sternotomy with the horizontal section of the sternum just below the second chondrosternal junction, subperiosteal division of the fourth, fifth, and sixth ribs just lateral to their costochondral junction on both sides, and bilateral section of the seventh, eighth, and ninth costal cartilages (Fig. 8). A Parham fracture band and silver wire were introduced beneath the sternum through the mesial ends of the fifth and sixth interspaces, and traction applied by the use of rubber bands attached to a special brace fitted on the patient. Ten months later the patient stated he was symptom-free, and the correction of the deformity had been maintained. The second case reported by Alexander was in a woman, 20 years of age, who two years previously was injured in an automobile accident. In this case the operation consisted of resection of the third, fourth, and fifth left costal cartilages and the fourth and fifth on the right side, and the body of the sternum from the level of the second to the sixth chondrosternal junctions (Fig. 9). According to Alexander, the technique for both operations was suited to the occasion and in both the purposes for which operation was performed were fulfilled.

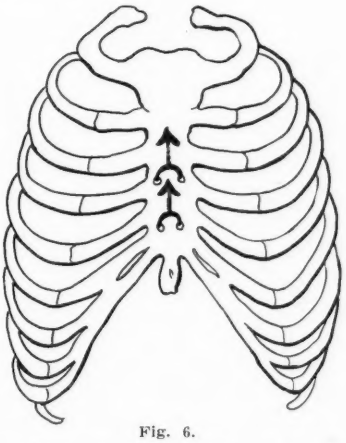


Fig. 6.

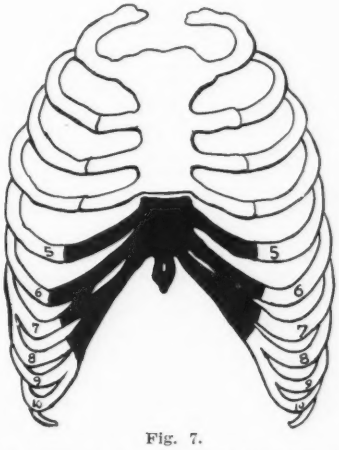


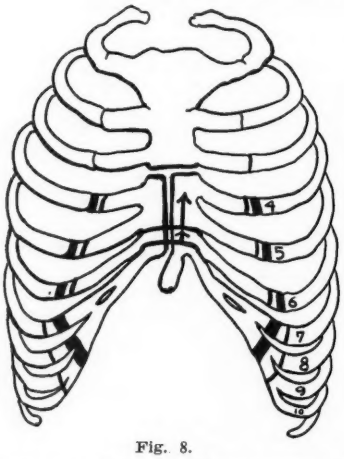
Fig. 7.

Fig. 6.—Diagram illustrating procedure performed by Zahradnicek,[34] 1925, consisting of introduction of two wires through perforations in the sternum and application of traction. (One case, successful.)

Fig. 7.—Diagram of operation performed by Lexer, reported by Hoffmeister,[35] 1927. Black area represents part resected. (One case, successful.)

Also in 1931 Ombrédanne reported the results in four cases operated upon by a procedure somewhat similar to that used by Alexander in his first case and stated that he first devised it in 1925. The operation

consisted of a T-shaped sternotomy with the horizontal bar of the T just below the level of the second chondrosternal junction, a cuneiform excision of a small section of the fourth to the seventh costal cartilages, inclusive, near the chondrocostal junction on both sides and the application of wire traction to the two sides of the sectioned sternum to hold them in the elevated position (Fig. 10). In association with Garnier[47] seven cases were operated upon in this manner, with three deaths and four satisfactory results. Two of the postoperative deaths occurred rather suddenly the evening following operation and are explained on the basis of torsion of the cardiac pedicle. The third death occurred five days after operation. A similar T-sternotomy procedure was employed by Martin[48] in one case with a satisfactory result, and Perrot[50] in two cases, one of which had a satisfactory result and the other ended fatally. Rocher and Laporte[39] also reported one case in which a T-sternotomy was performed but without section or cuneiform



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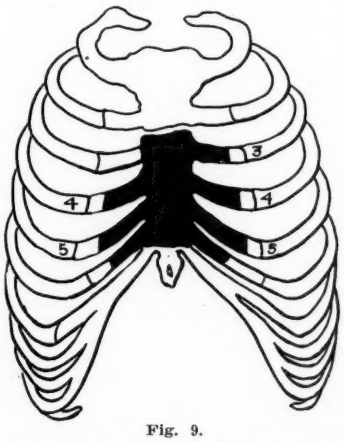

Fig. 9.

Fig. 8.—Diagram illustrating operation performed by Alexander,[38] 1931, in first case, consisting of a T-sternotomy and subperiosteal division of the fourth, fifth, and sixth ribs and the seventh, eighth, and ninth costal cartilages, bilaterally, as indicated by wide black area. The arrows represent traction. (One case, successful.)

Fig. 9.—Diagram of operation performed by Alexander,[38] 1931, in second case and subsequently performed by Truesdale,[55] 1938. Black area represents part resected. (Both cases, successful.)

excision of the costal cartilages (Fig. 11). Postoperatively the patient was very restless and the traction wire broke. The end result was unsatisfactory. Richard and Dupuis[51] also reported two cases in which the T-sternotomy procedure was employed with a satisfactory result in the first case and an unsatisfactory result in the second. In the latter case the procedure was slightly modified in that the adjacent costal cartilages were not sectioned, which according to these authors was a mistake. They also employed three tibial grafts to separate the sectioned edges of the sternum and prevent retraction. Because they were dissatisfied with the operative results and the tendency for the deformity to recur, Richard devised a method which they believe is a desirable pre-

and postoperative adjunct and may in some cases avert operation. The procedure consists of placing the patient in hyperextension and the application of a body cast with a window immediately over the deformity and epigastric region. A football bladder is placed between the anterior wall of the cast and the skin of the abdomen in the subumbilical region with the neck of the bladder coming through the cast to the outside. Thus, by inflating the bladder to the desirable degree, abdominal respiration is suppressed, the abdominal contents are forced upward, and the patient is forced to breathe with the lower thoracic portion.

In 1931 Sauerbruch stated that the procedure he first employed (Fig. 5) produced disturbances in respiration and for this reason he revised the procedure. He reported a case in which he performed an entirely different operation consisting of resection of a small segment of the fourth to the eighth costal cartilages inclusive on both sides (Fig. 12). A wire was placed around the body of the sternum and traction applied from an overhanging frame on the patient's bed. The patient was completely relieved. A somewhat similar procedure was performed by Mathieu[45] in a case reported in 1933 with a successful result; two other operations were subsequently performed by this author and included in Garnier's report.[47]

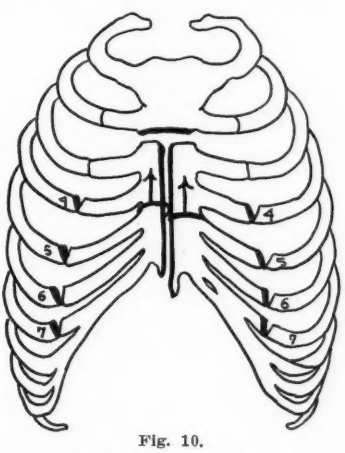

Fig. 10.

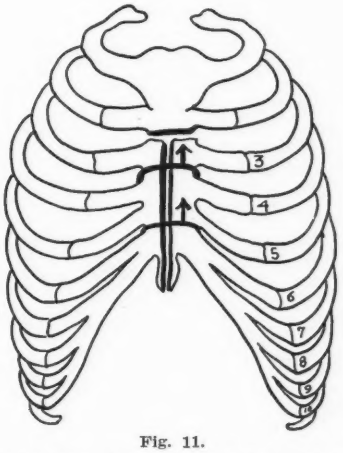

Fig. 11.

Fig. 10.—Diagram illustrating first type of operation performed by Ombrédanne,[50] 1931 (four successful, two fatal cases), and also by Garnier[47] (one fatal), Martin[46] (one successful), Perrot[50] (one successful, one fatal), and Richard and Depuis[51] (one successful, one unsatisfactory).

Fig. 11.—Diagram of procedure performed by Rocher and Laporte,[39] 1931, consisting of T-sternotomy and traction without costal cartilage division. (One case, unsatisfactory.)

Carr,[44] in 1933, reported a case in which operation was performed by Jerome Head. This consisted of resection of the third, fourth, fifth, sixth, and seventh costal cartilages on the left side, the fourth, fifth, sixth, and seventh costal cartilages on the right and the lower half of the body of the sternum including the xiphoid (Fig. 13). Although this case had a satisfactory result, Carr states that a second case in which

operation was performed by Head ended fatally. A similar procedure was performed by Truesdale[55] in a case he recently reported. On the twelfth postoperative day sudden massive hemorrhage occurred from the wound and post-mortem examination revealed a suppurative mediastinitis and an opening in the superior vena cava. Because of this complication, Truesdale performed a subperiosteal excision of the depressed portion of the sternum and costal cartilages and obtained a satisfactory result in a case which he had operated upon one year previously and which had recurred. The first operation performed in this case, which he previously reported,[54] consisted of resection of a portion of the third to the seventh costal cartilages, inclusive, on both sides near the costal margin, section of these costal cartilages near their sternal attachment, transverse section of the body of the sternum at the level of the third interspace, and the application of traction to the mobilized sternum.

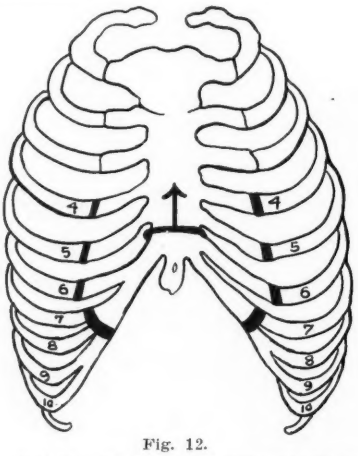

Fig. 12.

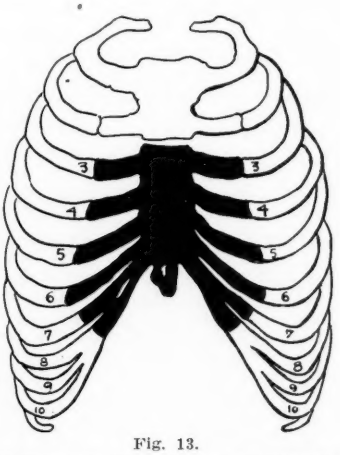

Fig. 13.

Fig. 12.—Diagram illustrating second type of procedure performed by Sauerbruch,[41] 1931, consisting of resection of a small segment of the fourth to the eighth costal cartilages, inclusive, on both sides and the application of wire traction to the sternum. (One case, successful.)

Fig. 13.—Diagram illustrating procedure performed by Head, 1933, and reported by Carr[51] and the second case operated upon by Truesdale.[55] (Head, one successful, one fatal case; Truesdale, one case fatal.)

In 1934 Garnier, who had joined Ombrédanne in 1931, after considerable operative experience with this condition presented the operative procedure which he and Ombrédanne considered the most rational and the safest. It is performed in two stages; the first stage consists of resection of approximately 2 cm. of the third to the eighth costal cartilages, inclusive, on one side. The second stage performed about one week later consisted of doing the same thing on the opposite side and a transverse section or cuneiform excision of the sternum at the level of the second interspace, thus permitting the depressed sternum to be easily mobilized (Fig. 14). A specially designed vise or "pince" is applied to the sternum at the fourth interspace to which wire traction

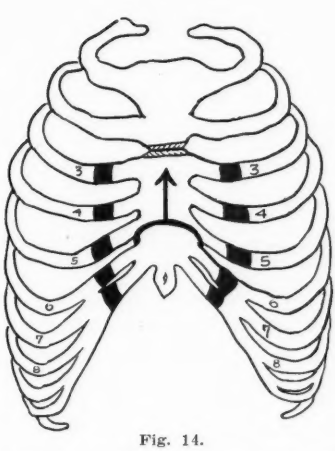

Fig. 14.

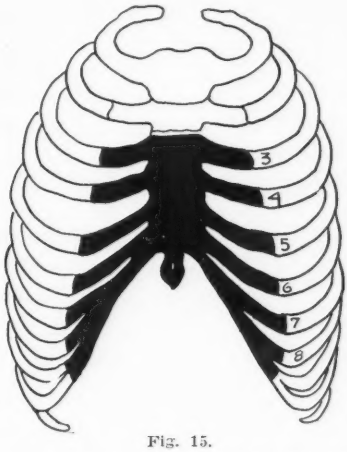

Fig. 15.

Fig. 14.—Diagram illustrating second type of procedure performed by Garnier,[47] 1934, and also by Mathieu,[45] 1933, Truesdale,[54] 1936 and the authors, 1937. Black area indicates part resected. A transverse cuneiform incision in the sternum is made at about the level of the second intercostal space and traction applied to the sternum, as represented by arrow. (Garnier, two cases satisfactory; Mathieu, three cases satisfactory; Truesdale, one case unsatisfactory; Ochsner and DeBakey, one case satisfactory.

Fig. 15.—Diagram illustrating one of three types of operative procedures that have been performed and may be termed *chondrosternal resection.* The black area represents part resected. (Sauerbruch, four cases; Lexer, one case; Alexander, one case; Head, two cases; Truesdale, two cases. Ten cases, two fatal.)

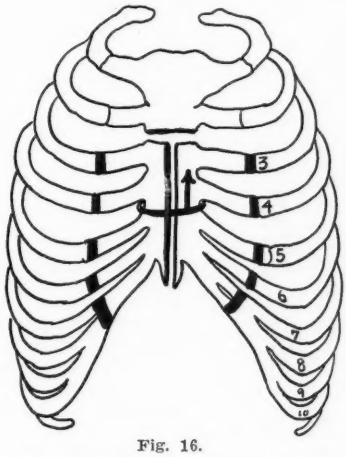

Fig. 16.

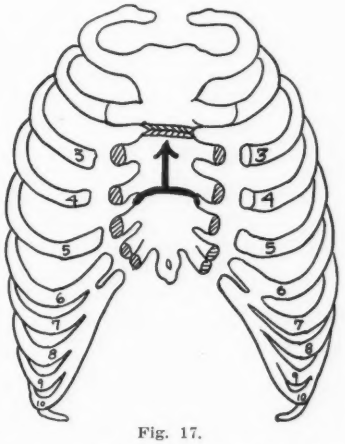

Fig. 17.

Fig. 16.—Diagram illustrating second type of operative procedure that has been performed for chōnē-chondrosternon, the *T-sternotomy* procedure, which was done in all cases, with or without costal cartilage division. The wide black bands indicate costal cartilage division or resection. In a few of the cases, however, this was not done. The arrow represents application of traction. (Ombrédanne, six cases; Garnier, one case; Martin, one case; Perrot, two cases; Rocher and Laporte, one case; Alexander, one case; Richard and Dupuis, two cases. Fourteen cases; four fatal, two unsatisfactory, eight successful.)

Fig. 17.—Diagram illustrating third type of operative procedure consisting of *sternal mobilization and chondral division or resection.* (Sauerbruch, one case; Garnier, two cases; Mathieu, three cases; Truesdale, one case; Ochsner and DeBakey, one case. Eight cases, one unsatisfactory.)

is applied by fixing the opposite end of the wire to a frame built into the anterior valve of a plaster cast previously fitted to the patient. Garnier states that they have performed two operations in this manner with very satisfactory results. The operation the authors performed in the present case with slight modification is similar to this latter procedure of Garnier.

In summarizing the thirty-two various operative procedures which have been devised and performed on thirty-one patients, it is observed that they may be classified into three groups. Parenthetically it may be stated that the procedures of Lexer and Zahradnicek are excluded from this group because of obvious lack of rational procedure in the former case and because of the marked departure from surgical principles in the latter. The three types of operative procedures are: (1) chondrosternal resections, (2) the T-sternotomy procedures with or without costal cartilage division and traction, and (3) sternal mobilization and costochondral division or resection.

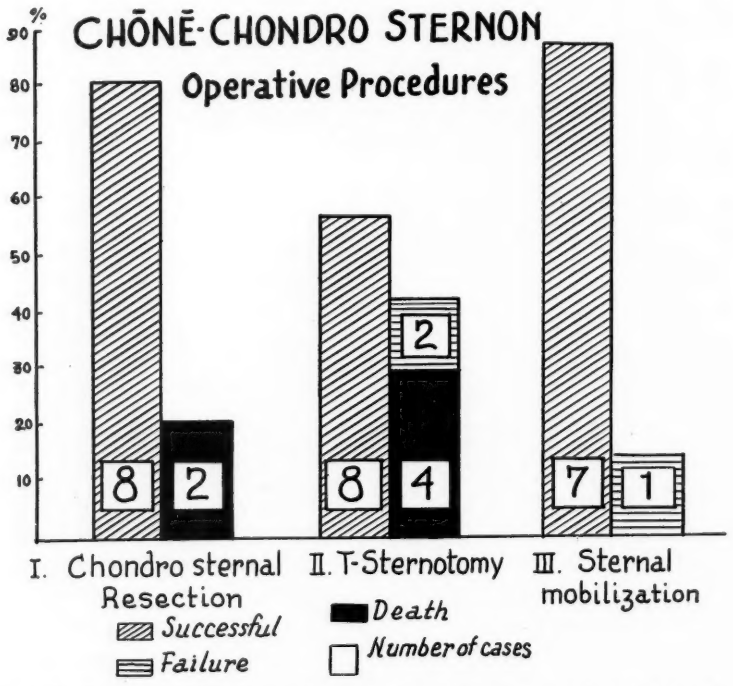

Chart I.—Results of operative procedures that have been performed in chōnē-chondrosternon in a total number of thirty-two cases.

Ten[28, 35, 38, 44, 55] of the thirty-two operations may be classified in the first group (Fig. 15). Of these ten cases there were eight (80 per cent) with satisfactory results and two deaths, a mortality of 20 per cent (Chart I). There were fourteen[38, 39, 40, 47, 48, 50, 51] cases in which

TABLE I

OUTLINE OF ALL CASES OPERATED UPON (COLLECTED FROM THE LITERATURE)

AUTHOR	DATE	AGE	SEX	ETIOLOGY	CLINICAL MANIFESTATIONS	PROCEDURE	RESULT	REMARKS
Meyer, L.[30]	1911	16	M	Congenital	Tuberculosis, marked dyspnea, heart displaced to left.	Resection 2.5 cm. rt. 2nd and 3rd costal cartilages.	Dyspnea relieved but unsatisfactory	
Sauerbruch[28]	1913	18	M	Congenital	Palpitation, dyspnea, heart displaced to left.	Resection 5th-9th costal cartilages and portion of ribs on left side adjacent part of sternum.	Satisfactory	
3 other cases						Same procedure.	Satisfactory	
Zahradnicek[34]	1925	16	M	Congenital	Palpitation, dyspnea.	Perforation of sternum and traction by wire.	Satisfactory	
Hoffmeister[35] (Lexer)	1927	19	M	Congenital	Palpitation, dyspnea, precordial pain.	Resection of sternum from level of 5th rib above depression and adjacent costal cartilage 5th-9th, bilaterally.	Satisfactory	
Alexander[38]	1931	16	M	Trauma	Dyspnea, pain, dysphagia.	T-sternotomy. Section of 4th-9th ribs and cartilage, bilaterally; traction.	Satisfactory	Satisfactory. 10 months later.
Ombrédanne[40]	1931	7	F	Congenital	Dyspnea, debility.	T-sternotomy; traction.	Satisfactory	
		5	F	Congenital	Dyspnea, debility.	T-sternotomy; traction.	Satisfactory	
		17	M	Congenital	Dyspnea, debility. Shallow respiration.	T-sternotomy; traction.	Satisfactory	
		28	M	Congenital	Precordial pain, palpitation.		Fatal	Died suddenly 48 hours after operation. Suggests torsion of heart pedicle as cause.

TABLE I—CONT'D

AUTHOR	DATE	AGE	SEX	ETIOLOGY	CLINICAL MANIFESTATIONS	PROCEDURE	RESULT	REMARKS
Rocher and Laporte[39]	1931	12	M	Congenital		T-sternotomy; traction.	Unsatisfactory	Immediately after operation child nervous and restless; traction broke.
Sauerbruch[41]	1931		F	Congenital		Section of 4th to 8th costal cartilages both sides with traction on sternum.	Satisfactory	
Carr (Head)[44]	1933	19	F	Congenital	Dyspnea on exertion, palpitation, heart displaced to left.	Resection 3, 4, 5, 6, 7 costal cartilages left and 4, 5, 6, 7, rt.; lower half sternum and xiphoid.	Satisfactory	
(Head)							Fatal	Died 48 hours after operation.
Mathieu[45]	1933	17	F	Congenital	Precordial pain, dyspnea.	Resection costal cartilage bilaterally except first two; traction.	Satisfactory	
Two others reported by Garnier[47]		20	F	Congenital	Precordial pain, dyspnea.	Same procedure.	Satisfactory	
		20	F	Congenital	Precordial pain, dyspnea.	Same procedure.	Satisfactory	
Garnier[47]	1934	Child		Congenital	Palpitation, respiratory embarrassment.	Transverse section of sternum below level of 2nd rib. Resection 2 cm. costal cartilage both sides. Traction.	Satisfactory	
		10	M	Congenital		Same procedure.	Satisfactory	
		16	M	Congenital		T-sternotomy.	Fatal	Died suddenly evening of the operation.
		6½	F	Congenital		T-sternotomy.	Fatal	Died five days after operation.
		12	F	Congenital	Tuberculosis.	T-sternotomy.	Satisfactory	

TABLE I—CONT'D

Martin[48]	1934	Child	M	Congenital	Respiratory embarrassment.	T-sternotomy.	Satisfactory	
Perrot[50]	1934	18	M	Congenital	Marked dyspnea and pain.	T-sternotomy.	Fatal	Death occurred suddenly 36 hours after operation.
Richard and Depuis[51]	1934	9	M	Congenital	Dyspnea, poor peripheral circulation.	T-sternotomy and section of 3rd to 5th costal cartilages; traction.	Satisfactory	Pleura opened, postoperative emphysema requiring multiple incisions.
		5	M	Congenital		T-sternotomy with bone graft and no chondro-costal section or traction.	Unsatisfactory	
Truesdale[54]	1936	17	M	Congenital	Dysphagia, nausea, anorexia.	Section 3, 4, 5, 6 costal cartilages bilaterally at costal and sternal margins. Transverse section sternum at 3rd interspace. Traction.	Satisfactory	Condition recurred 1 yr. later. Second operation performed. Resection of body of sternum. Result satisfactory.
Truesdale[55]	1938	31	M	Congenital	Pain left chest. Epigastric distress.	Resection body of sternum and 3, 4, 5, 6, 7 costal cartilages.	Fatal	Massive hemorrhage from wound 12th postoperative day due to opening in vena cava. Suppurative mediastinitis.

the T-sternotomy procedure with or without costal cartilage division was used (Fig. 16) with eight (57.1 per cent) successful results, two (14.2 per cent) unsatisfactory results, and four (28.5 per cent) deaths (Chart I). There were eight ([41, 45, 47, 55] authors') cases in which costochondral division or resection with sternal mobilization and traction was employed with no deaths and only one (12.5 per cent) unsatisfactory result (Fig. 17, Chart I). A critical analysis of these three types of procedure clearly reveals the definite superiority of the latter type and is strikingly illustrated in Graph I. There were no fatalities in this group as compared to a mortality rate of 20 per cent in the first and 28.5 per cent in the second group and the incidence of satisfactory results is higher than in both other groups (Graph I). Moreover it may be considered a more rational procedure because it consists of repairing a structural defect without disturbing physiologic function.

The first procedure consisting of chondrosternal resection to varying degrees cannot be considered rational because although the structural defect is removed the physiology of respiration is undoubtedly disturbed as had been observed by Sauerbruch[41] and Cutler.[142] Actually it consists of substituting one disturbance for another which in some instances may be even worse. The T-sternotomy procedure has the disadvantages of being extensive, complicated, and likely to recur. For these reasons the authors are of the opinion that the procedure of costochondral division or resection with sternal mobilization and traction is the operation of choice in chōnē-chondrosternon.

CASE REPORT

Chief Complaint.—Shortness of breath, pain in chest.

Present Illness.—Patient stated that until 10 years of age, she was considered a normal child. At this age there was noticed a beginning depression in the region of the lower portion of the sternum. This "sinking" in of the sternum gradually progressed with the growth of the patient but during the past year has rapidly become worse. There has been a weakness in the right shoulder for two years, during which time there seemed to be a tendency for it to drop lower than the opposite shoulder. Simultaneously with the development of the depression in the sternum the patient began to become dyspneic on slight exertion. Associated with this, there is a sensation of tightness and constriction in the chest and the patient is always conscious of her heart beating. For the past six months, the dull aching pain located in the precordium and somewhat to the right of the depression has become progressively worse, and the dyspnea has increased in severity. Occasionally, she has attacks of epigastric distress, dysphagia, and vomiting. She also complains that her hands have a tendency to become cold and blue, particularly during cold weather; however, there is no redness or pain. The hands are usually cold and moist. She has been observed by a number of physicians who have prescribed breathing and postural exercises and various orthopedic measures with no relief of symptoms.

Past History.—The patient's health was good until 10 years of age. There were no rickets and no serious debilitating diseases. Frequent attacks of tonsillitis occurred during childhood, also varicella. There were no operations.

Family History.—The father and mother are living and in good health. Father is slender and over 6 feet in height. Mother is of average height and size. Two

brothers are tall, slender, over 6 feet in height, and in good health. Three sisters all tall and slender, the shortest being 5 feet 8½ inches in height. There is no evidence of heart disease, tuberculosis, or syphilis in the family. One aunt died of carcinoma of the breast. No member of the family either on the mother's or the father's side is known to have the patient's deformity.

Menstrual History.—Began menstruation at 13 years of age. Periods are regular, twenty-eight days apart, and usually last from eight to ten days. Three years ago, she began to have dysmenorrhea. This is associated with considerable nausea and vomiting.

Physical Examination.—Physical examination revealed a white female, 21 years of age, of asthenic habitus, 6 feet in height, and weighing 113 pounds, in apparently fairly good health with an obvious and marked deformity, chōnē-chondrosternon, of the anterior chest wall. The temperature was normal, pulse 76 per minute, and blood pressure 110/60. The only significant findings were those of the thorax. The chest is extremely long and there was a marked depression in the sternochondral region beginning just below the junction of the body of the sternum and the manubrium and extending caudad to the xiphoid measuring approximately 18 cm. (Fig. 18). The lateral edges of this depression were formed by a sharp dorsal angulation of the costal cartilages and ribs at the costochondral junction and a ventral angulation at the chondrosternal junction. This angulation was more obtuse on the left side and more acute on the right so that the sternum appeared to be

Fig. 18.—Photograph of authors' case before operation, showing marked depression in the chondrosternal region and the slightly exaggerated dorsal kyphotic curve.

turned in its longitudinal axis with the anterior surface slightly facing the right side. The depression measured approximately 11 cm. in width and 7.5 cm. in depth. The left thorax was slightly larger than the right measuring on the left side 38 cm. in circumference from the midline posteriorly to the midline anteriorly at the level of the third rib anteriorly and 32 cm. in circumference at the corresponding level on the right side. There was a 4 cm. chest expansion from expiration to deep inspiration. There was a slight scoliosis with the convexity towards the left side. Slight tenderness was present over the third, fourth, and fifth costal cartilages on the right side. The lungs were resonant and no râles were present. The heart was dis-

placed to the left with the left border in the anterior axillary line. There were no murmurs. Examination of the abdomen revealed no abnormalities. The extremities were unusually long (Fig. 2), and the fingers displayed characteristic arachnodactylia. The reflexes were normal.

Fig. 19.—Photograph showing method for the maintenance of traction on the mobilized sternum after operation. The cast which had been applied to the patient previous to operation and had been bivalved in the axillary line has a rigid metal frame in the form of a U securely applied to the anterior valve of the cast, bridging over the window in the chondrosternal and epigastric regions. The rubber bands exert traction and are attached to the metal frame above and to the Parham-Martin band passing around the sternum below.

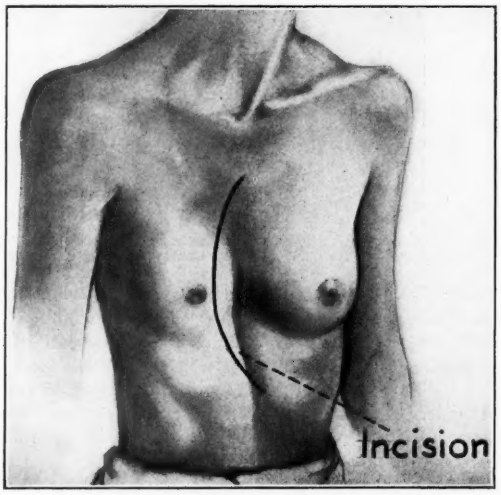

Fig. 20.—Drawing illustrating type of incision employed in authors' case.

Laboratory Examinations.—Erythrocyte count, 5,230,000 per c.mm.; hemoglobin, 90 per cent; leucocyte count, 8,000 per c.mm. with 66 per cent neutrophiles, 30 per cent small lymphocytes, 3 per cent large lymphocytes, and 3 per cent eosinophiles. Urinalysis and blood chemistry revealed no abnormalities.

Roentgenologic examination in the anteroposterior view showed no evidence of disease in the lung fields. The heart was normal in size and shape but located entirely to the left of the midline. There was a slight lateral curvature of the dorsal vertebrae with the convexity toward the left side. In the lateral view the sternum appears markedly depressed so that the posterior surface is about 2.5 cm. from the anterior margin of the eighth and ninth vertebral bodies (Fig. 3).

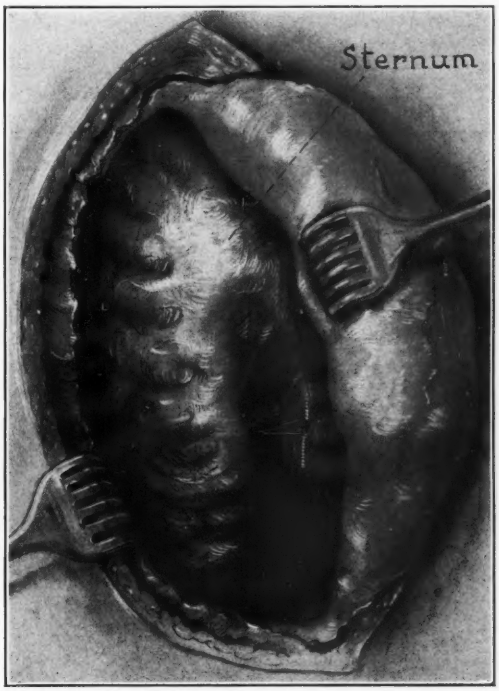

Fig. 21.—Drawing showing mobilization of skin flaps and attachment of pectoral muscles so as to expose entire anterior surface of sternum and adjacent costal cartilages.

Before operation a plaster of Paris cast was placed around the body and moulded to fit perfectly about the shoulders and the hips, and a window was cut in the anterior wall of the cast so as to permit the entire chondrosternal and epigastric regions to be exposed. Immediately over the window and at the level of the fifth anterior intercostal space an iron bar bent in the form of an inverted U was securely applied to the cast so as to bridge over the window in cast which will be used to maintain traction after the operation (Fig. 19). The cast was then bivalved in the axillary line. Operation was performed on May 23, 1937. Ethylene gas-cyclopropane anesthesia was employed. A semilunar incision beginning at the second left chondrosternal junction and extending caudad with the convexity to the right side about 5 cm. below the ensiform cartilage was made (Fig. 20). The skin flaps were mobilized on both sides including the pectoral muscle attachments, thus exposing the entire anterior surface of the sternum and adjacent costal cartilages (Fig. 21).

About 2 cm. of each costal cartilage from the third to the ninth costal cartilages on the right side were then resected subperiosteally about 2 cm. from the chondrosternal junctions (Fig. 22). The sternum was still unyielding. The attachment of the recti muscles below and the diaphragm posteriorly were severed, and the resection of the costal cartilages was done on the left side similar to that on the right. However, even after the entire body of the sternum including the xiphoid was freed of its chondral and muscular attachments it could be mobilized anteriorly only with considerable difficulty due to the fibrous tissue attachment to its posterior surface (Figs. 23 and 24). It was observed at this time that the distance between posterior surface of the sternum and the anterior surface of the bodies of the eighth and ninth ribs was only about 2 cm. A transverse cuneiform osteotomy was then made in the sternum at the level of the third chondrosternal junction which overcame the tendency of the sternum to retract to its original position. A Parham-Martin band bent in the form of a U was placed around the sternum between the fifth and sixth chondrosternal junctions. The right end of the band was brought out

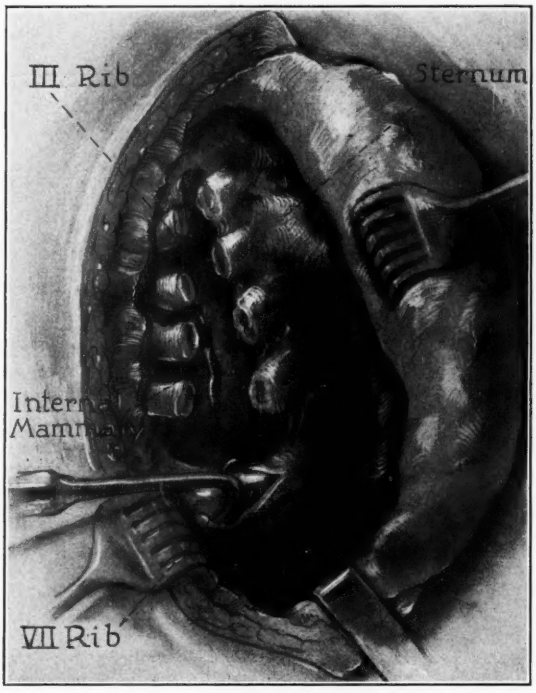

Fig. 22.—Drawing made at time of operation in authors' case, illustrating resection of costal cartilages.

through the incision and the left end through a small stab incision in the flap (Fig. 25). The wound was closed by first suturing the pectoral muscles to the periosteum on the lateral margins of the sternum on both sides with interrupted silk and approximating the subcutaneous fascia and skin edges. A small rubber dam drain was placed in the cavity beneath the sternum formed by the mobilization of the sternum anteriorly. After applying sterile dressings to the wound the anterior valve of the cast was placed in position and rubber bands were attached to the

two ends of the Parham-Martin band and fixed to the overhanging metal frame (Fig. 19). The rubber bands were found to exert sufficient traction to hold the sternum securely in its new position. The anterior valve of the cast was then securely attached to the posterior valve which had remained in position during the operation. Immediately after operation the patient was given 500 c.c. of unmodified blood and left the operating room in excellent condition.

Progress.—May 24, 1937, the day following operation, the patient was very nauseated; this was found to be due to the morphine because as soon as its administration was discontinued, the nausea and vomiting ceased. Her general condition remained very good.

Fig. 23.—Drawing made at time of operation in authors' case, illustrating mobilization of sternum and fibrous tissue attachment to its posterior surface.

On May 29, 1937, the posterior valve of the cast was removed and wide adhesive strips were used to keep the anterior valve securely in position; following this the patient was much more comfortable. Attention is directed to the fact that the anterior valve was actually supported above by resting on the anterior surface of the shoulders and below on the anterior superior iliac spines as shown in Fig. 19.

On June 2, 1937, sutures were removed, the wound healing per primam. There is still a slight amount of serous drainage where the drain was inserted.

The patient was in a chair and quite comfortable on June 5, 1937. Roentgenologic examination in the lateral view revealed the Parham-Martin band passing around the sternum which has been mobilized anteriorly (Fig. 26).

Patient was up and walking about on June 10, 1937.

On the thirty-fifth post-operative day, June 27, the Parham-Martin band was removed by cutting the right projection at the skin margin and placing traction on the left. No discomfort was experienced by the patient. The sternum appeared fixed in its new position.

Fig. 24.—Photograph made at time of operation in authors' case showing difficulty in mobilizing sternum even after resection of costal cartilages, due to fibrous attachment along the posterior surface of the sternum.

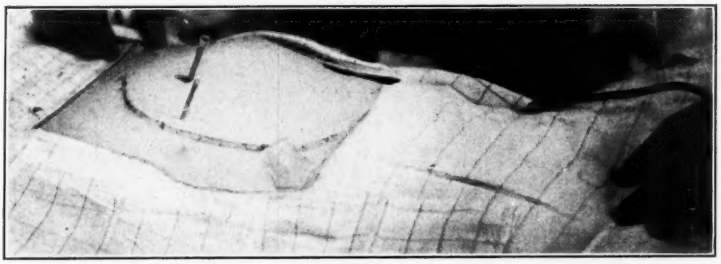

Fig. 25.—Photograph made after termination of operation in authors' case, showing two ends of Parham-Martin band which have been brought out to the surface and to which traction will be applied.

The patient was discharged from the hospital, June 30, 1937. She has been observed several times since operation and on each occasion has stated that the improvement has been maintained. The most recent observation was made on April 2, 1938, almost one year since the operation. The patient has been completely relieved of all previous complaints of dyspnea, palpitation, and precordial pain. She has gained 7 pounds in weight, and feels much stronger. The correction of the thoracic deformity also has been maintained (Fig. 27). It was also observed that chest expansion which was formerly 4 cm. from expiration to deep inspiration has been increased to 6 cm. Roentgenologic examination in the lateral view shows the sternum to be in a more normal position (Fig. 28).

Fig. 26.—Roentgenogram made approximately two weeks after operation, showing a Parham-Martin band passing around the sternum which has been mobilized anteriorly.

Fig. 27. Fig. 28.

Fig. 27.—Photograph of patient made on April 2, 1938, almost one year after operation, showing correction of chōnē-chondrosternon.

Fig. 28.—Roentgenogram of chest in lateral view of authors' case, made on April 2, 1938. The white outlined area reveals position of sternum and maintenance of correction of deformity.

SUMMARY

1. Chōnē-chondrosternon is considered preferable to the previously used terms (funnel chest, pectus excavatum, koilosternia, trichterbrust, thorax en entonnoir) describing the thoracic deformity which may be defined as a usually congenital and rarely acquired depression of the lower portion of the sternum and the adjacent costal cartilages which characteristically becomes progressively more marked as the individual grows.

2. A complete review of the literature is made from the standpoint of etiology and pathogenesis which may be classified into congenital and acquired factors. The most prominent characteristics of the former are: (1) heredity, (2) growth disturbances (sternal developmental arrests, costal overgrowth, delayed sternal ossification, muscle contracture, and hyperpituitarism), (3) mediastinitis, (4) mechanical intrauterine compression, and (5) syphilis. The acquired factors consist of: (1) rickets, (2) trauma, and (3) faulty posture.

3. Chōnē-chondrosternon was found to occur in 0.059 per cent of a total of 46,705 persons examined. The sex incidence was found to be 78.1 per cent males and 21.8 per cent females in 268 collected cases.

4. The anatomic, pathologic character of chōnē-chondrosternon consists of depression of the sternum and slight rotation on its longitudinal axis usually to the right, a decreased anteroposterior thoracic diameter, and an increase in the transverse thoracic diameter. There is usually lateral displacement of the heart to the left as well as compression and lateral rotation. The lungs and the esophagus may also be compressed, and the diaphragm is usually depressed.

5. There are frequently no subjective or objective clinical manifestations. However, when they are present, these manifestations consist of cardiac disturbances such as dyspnea, palpitation, precordial pain, and in rare instances decompensation; pulmonary disturbances such as dyspnea, cyanosis, cough, and diminished vital capacity; and digestive disturbances such as dysphagia and dyspepsia. In general, these patients are of asthenic habitus and usually display characteristic weakness and debility. Not infrequently, they have poor resistance to infection and a tendency toward bronchitis and tuberculosis.

6. Treatment may be divided into conservative and operative. The conservative methods consist of breathing and postural exercises and orthopedic measures.

7. The various types of operative procedures that have been performed are reviewed and analyzed and may be classified into three groups: (1) chondrosternal resections, (2) T-sternotomy with or without costal cartilage division and traction, and (3) sternal mobilization and costochondronal division or resection. There have been ten operations performed according to the first group, with eight (80 per cent) satisfactory results and two (20 per cent) deaths; there were fourteen

cases performed according to the second group with eight (57.1 per cent) successful results, two (14.2 per cent) unsatisfactory results and four (28.5 per cent) deaths; there were eight cases performed according to the third group with seven (87.51 per cent) successful results, one (12.5 per cent) unsatisfactory result, and no deaths.

8. A case is reported in detail with a description of the operative procedure employed. The patient examined approximately one year after operation revealed that the correction of the thoracic deformity had been maintained and the symptoms completely relieved.

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DISCUSSION

DR. JEROME R, HEAD, Chicago, Ill.—As Dr. Ochsner has said, I have operated upon two patients with pectus excavatum. One patient was relieved of her symptoms, the other died following the operation. The operation which I have performed (removal of the depressed lower sternum and cartilages) appears to me to be a less satisfactory procedure than that described by Dr. Ochsner. Certainly when I have another case to do I shall use this technique.

If one removes the sternum and cartilages the resection must be wide and the permanent defect in the anterior chest wall correspondingly extensive. As time goes on, the ribs, which have been deprived of their sternal attachments, tend to bend inward with the result that pressure on the heart recurs.

The patient upon whom I performed this operation was relieved immediately and has since resumed and continued her work as a nurse. At the beginning of the operation her pulse was 120 per minute. As soon as the sternum was freed and elevated it fell to 90 and throughout the postoperative period was lower than it had been before the operation.

It is my impression that the presence or absence of symptoms in cases of pectus excavatum depends largely upon the success with which the heart has escaped to the left. In this case it had not escaped at all but lay in its normal position imprisoned between the sternum and the spine.

The second case upon which I operated was a man 60 years old who had developed cardiac decompensation. Dr. James Carr was the cardiologist in this case. We both felt that constriction of the heart was an important factor in the intractable decompensation and that if more space could be afforded, improvement might follow. The patient was too ill to permit an extensive operation, and for this reason I attempted merely to lengthen the ribs over the heart by severing them and interposing bridges of Lane plates. During the operation the pleura was accidentally opened and a pneumothorax produced. The patient became cyanotic and died on the operating table.

DR. CALVIN SANDISON, Atlanta, Ga.—It is very kind of Dr. Ochsner to invite a guest to discuss his paper. I think it is fine that he has called attention once more to a satisfactory way of handling a most difficult problem, especially a problem in which patients are completely decompensated or bedridden and need help very badly.

In our work here, Dr. Lawson Thornton and I have had two similar experiences. Our first patient, a girl of 13 years, fulfilled all of the clinical manifestations Dr. Ochsner mentioned, including cyanosis and dyspnea, and she was unable to be out of bed. In the lateral x-ray of the chest the sternum almost touched the vertebrae. We did the T-shaped type of operation, mentioned by Dr. Ochsner, including osteotomy of five ribs on each side below the third rib. We followed the plan of operation described by Dr. John Alexander.

This was practically the same type of deformity shown by Dr. Ochsner. The cut edges of the sternum were held forward with rubber bands connecting with a Parham band around these sternal edges and attached to a metal arch imbedded in a body plaster cast.

Dr. Alexander called attention to the way in which the Parham band should be applied. He has told us that he had his band too high, and the upper angle of the sternum, with movements of respiration, cut through the skin and formed an abscess which was alarming for a time, but the child recovered and was relieved of the symptoms from the cardiac compression.

Our first patient is now well and active. With the second patient, a boy of 14 years, we decided to use a different procedure. The T-incision was made without osteotomy of the ribs, the substernal tissues were dissected laterally and a large bone graft from the tibia was inserted between the separated sternal edges and wired in place. This method seems to us to be the most practical one. It provides an internal fixation of the bones; no external apparatus is necessary. The boy, like the girl, is now well and active.

DR. HAROLD BRUNN, San Francisco, Calif.—I have to report two patients who were operated upon at the Thoracic Clinic, University of California.

A boy of 6 years came to us because of a bronchiectasis involving both the upper and lower lobes of the right lung. He was a puny, anemic youngster and

showed a very marked deformity of his sternum with the heart on the right side, probably not congenital but the result of the pectus excavatum. He had frequent bouts of temperature which kept him in bed a considerable part of the time and he constantly coughed up large quantities of sputum. We thought it best in this case to rectify the sternal condition before attempting an operation for the bronchiectasis. In this case the sternum and the cartilages were removed and no attempt made to elevate it according to the method suggested by Dr. Ochsner.

The heart has not gone back into place but the boy's general condition has improved greatly and he is now ready for the operation of pneumonectomy which will be done sometime this summer. Pictures were taken of the patient before and after the operation showing a great change in his physical appearance.

The other case was that of a young woman who had had the deformity since childhood but had symptoms only in recent years so that at the time she entered our clinic she was unable to do housework and spent most of her time resting. She was a tall, slender, anemic looking person, very dragged and stooped and anoxemic, with some dyspnea. In her case the sternum was again removed completely with the cartilages by resection. She has not improved as much as the boy and I think the earlier the operation is done the better the result obtained. After physiotherapy, however, she has improved.

I wish to draw attention to the ligamentous band which is found under the sternum and many times the mediastinal adhesions to the pericardium which have to be removed in order to make the operation a success. I would like to say also that the deformity is not particularly great after the removal of the lower part of the sternum and cartilages. There is no considerable bulging and I am not as yet convinced that resection is not just as successful as other types of more complicated operations.

DR. RICHARD H. MEADE, JR., Philadelphia, Pa.—Dr. Ochsner has called attention to the fact that there are many cases with very marked deformity that never have any symptoms. Recently at the Episcopal Hospital in Philadelphia a patient was admitted because of exophthalmic goiter. It was seen she had a marked funnel chest and one wondered how she had gone 38 years without any symptoms whatsoever.

X-ray photographs of her chest, after outlining the esophagus with barium, showed the esophagus and heart displaced entirely to the left.

In the lateral view the sternum is shown almost in contact with the vertebrae, yet this patient has had absolutely no symptoms over a period of 38 years.

DR. T. C. DAVISON, Atlanta, Ga.—I have a patient under observation now, whom I have had since childhood. She is in her late twenties and has recently married, and I was wondering what effect pregnancy might have on this deformity. She has had no symptoms so far.

I would also like to ask Dr. Ochsner, in closing, to explain how the sternum, when it is elevated, will become reattached to the ribs after he has resected the cartilages and if it becomes attached satisfactorily.

DR. I. A. BIGGER, Richmond, Va.—I wish to ask Dr. Ochsner a question in regard to the advantages of overhead and plaster cast traction for keeping the sternum elevated. I noticed that the two pictures shown by Dr. Ochsner and Dr. Sandison both showed the use of the plaster cast.

In a case which I had in January, 1937, with very extensive injury to the thoracic wall, with a fracture of both clavicles, the ribs on both sides, and with marked depression of the breast plate, we passed a heavy silver wire around the sternum and then used the Balkan frame with overhead traction. I am sure a plaster cast has many advantages, but it seems to me that the Balkan frame may also have certain advantages. My patient was able to move around freely, sitting or reclining as he chose. Is such freedom of motion possible when plaster is used?

DR. ALTON OCHSNER, New Orleans, La.—I am indeed grateful to all the discussers of the paper.

Dr. Head referred to the cardiac manifestation. I think that displacement and fixation of the heart are always potential causes of clinical manifestations. There are two such cases reported, one of which ended fatally and one in which the patient's life was saved by the prompt removal of pleural fluid on the left side. These individuals had no symptoms whatsoever until they developed a pleural exudate, and because of the fact that the heart couldn't be shoved to the right on account of the lessening of the sterno-vertebral diameter, the heart was compressed producing very dangerous symptoms. The one patient died as a result of cardiac compression.

I think Dr. Sandison deserves a great deal of credit for the successful result which he obtained by means of a bone graft. As I said, it had been done in other cases but unsuccessfully, probably because the graft wasn't done by an orthopedist. The advantage of using the bone graft is that external fixation is not necessary. I would like to suggest if one uses the technique which we have used, that he use two Parham-Martin bands instead of one, because in our case the sternum broke at the lower end in very much the same way that one of John Alexander's cases gave way, at the manubrial sternal junction. If I have another one I plan to use two bands, distributing the tension on the sternum throughout its length.

I do not know what effect pregnancy might have upon one of these patients. I can imagine, though, that a person might have enough cardial reserve to carry on normally, but at the same time the added strain during pregnancy might produce manifestations. I think one might seriously consider doing something in such a case. I think the thing to do is to determine what her vital capacity is. If her vital capacity is much decreased I would suggest correcting the deformity.

We mobilized the costal cartilages on either side, and because of the increased length of the costal cartilages we did, as much as we could, a subperichondrial resection of the costal cartilage. I think we left enough perichondrium behind so that the costal cartilages reformed which can be palpated. I think that is the reason the sternum has become fixed in the corrected position.

The length of time which one should keep traction, I think, varies. This was a relatively young individual. We maintained traction for five weeks.

One reason why I prefer the plaster fixation is that these patients can be up and about. Our patient was up after the tenth day and was up and about for approximately four weeks, whereas if we had used any overhead traction she would necessarily have had to have been confined to her bed. I think that just as the osseous graft has probably an advantage over the plaster fixation, the plaster fixation is definitely better than overhead traction.

CINEFLUOROSCOPIC STUDIES OF PECULIAR BREATHING AND CHEST MOTION

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THROUGH the courtesy and kindness of Dr. William H. Stewart we are enabled to demonstrate some peculiar breathing noticed in some of our patients at the Lenox Hill Hospital, New York City. We are able to show the movement of the chest, diaphragm, and lungs through cinefluoroscopic reels that Dr. Stewart has prepared. He has called your attention to the great improvement he has made in the present moving pictures over those taken two years ago. They have been greatly slowed down, and it is much easier to study them now than in those in which the breathing was so accelerated.

The great advantage of course of these cinefluoroscopic films is that we have a permanent record of the movement of the chest and the organs for reference and comparative study. The great field we believe for the use of these films will be in teaching, where the normal and abnormal respiratory and cardiac movement can be graphically and repeatedly demonstrated; of course there also will be an advantage in them in having a permanent and convenient record of the movements shown for reference in the individual cases.

TECHNIQUE

Cinefluorography is a method of roentgenographic examination in which the image produced on a fluoroscopic screen is recorded on moving picture films. When these films are printed and projected they reproduce the visual effect originally present in the fluoroscopic screen and can be demonstrated to large groups in classrooms or auditoriums.

The idea for this method is almost as old as the x-ray. It is only recently, however, that technical advances have permitted the production of x-ray tubes and fluoroscopic screens efficient enough to produce a satisfactory image on the screen and photographic lenses and films sufficiently sensitive to record them clearly.

The actual technique of the examination can be best understood by considering the set-up used in an ordinary fluoroscopy. Instead of an observer sitting before the screen, we have a moving picture camera mounted in such a position as to cover all or part of the screen and record the motion of the shadows which we wish to study.

The camera used is an ordinary Bell and Howell 70 D Filmo. A special speed regulating device has been installed to permit exposures at 4, 8, 12, 16, and 24 frames per second. The lens on the camera is a Zeiss Biotar F 0.85. This lens is the fastest obtainable consistent with clear reproduction of the image. The films which we are now using are Agfa Superpan Supreme or Eastman XXX negative film which are exceptionally fast. They are especially sensitive to the light emanated

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by our fluoroscopic screen which was developed by Dr. Levy and Mr. West of London, England, and emits a yellow-green light. The film in the camera is shielded from exposure by the direct x-rays by a piece of lead glass mounted in front of the fluoroscopic screen. In order to produce an image of sufficient brightness for recording by this method, high x-ray currents must be employed. A General Electric 10 R.W. tube is used and exposures of from 5 to 10 seconds are made at a potential of about 100 kv. and a current of 100 ma. Such an exposure permits the recording of more than one cycle of cardiac or respiratory motion. For studies of thoracic conditions exposures may be made at 16 frames per second; thus in 5 seconds an 80 frame run can be obtained.

After the film has been exposed it is developed for about 10 minutes in x-ray developer in complete darkness and then fixed in x-ray fixing solution. The film is washed and dried in the ordinary manner. A positive print is made from this original film and when this has been developed and dried the ends are joined together to make a loop. From this intermediate loop any desired number of feet of negative print can be made for projection and study.

It is not necessary to describe the factors responsible for the effortless normal act of respiration, in which the two sides of the chest rise symmetrically with inspiration, and fall with expiration—the two halves of the diaphragm contracting equally with the former and rising with their relaxation with the latter. We do wish to emphasize, however, even though it may seem trite, that if one or more of these relating physiologic factors are affected by disease or therapy, we may throw the symmetry out of balance and cause bizarre and unusual respiratory movement.

Many observers appear to feel that the collapsed lung after pneumothorax, with or without phrenic crushing, is almost completely at rest; simple observation shows that this is not true in general. This is shown by our case of Mr. P.

Roentgenograms have shown that a collapsed lung expands somewhat on inspiration and contracts on expiration; this movement will vary greatly in different individuals. It is influenced by many factors, namely, intrapleural pressure, diaphragmatic movement, expansibility of the particular lung, flexibility of the thoracic wall, and a patency of the bronchi, among others. This is not of particular moment in therapy except that in some cases the artificial collapse affects the function of the lung considerably more than in others.

If, for example, the fixed foundation of one side of the chest is removed by the resection of ribs with a mobile mediastinum a curious and possibly serious breathing may result; if the heart is considerably displaced it may be affected and a varying degree of cyanosis and anoxemia may result. If the diaphragm has been paralyzed through a phrenic operation, if associated with a pneumothorax, and if adhesions are not present, a paradoxical movement is the rule.

The flexibility of the mediastinum may be noted from a slight degree to that of a pendulum movement with respiration, or mediastinal "flutter," or it may be more localized and herniate toward the opposite side

in a pneumothorax. When this occurs, of course, it frequently prevents a satisfactory collapse and we are unable to close successfully the lesion under treatment. It prevents our getting useful intrapleural interpretations on our manometer and often results in a further effort being made to fix the mediastinum with the introduction of a small amount of oil, or it may even cause a cessation of the pneumothorax therapy.

The mediastinum is that area in the chest that lies between the two pleuras and contains all the thoracic organs except the lungs. It is not necessary in this discussion to define its boundaries or various parts; suffice it to say that its contents are more or less movable containing, as it does, the heart, pericardium, blood vessels, lymphatics, nerves, and loose areolar tissue. In the healthy person there is little, if any, movement of the mediastinum and its contents with the respiratory act, but if various influencing factors are developed this fixability may be more or less affected and may bear a very considerable effect on the prognosis and course of the person's health or recovery. The weakest portions of the mediastinum are the anterior superior and posterior inferior parts which are therefore the most vulnerable and usually are the portions which show the effect of the influencing factors referred to above. The mediastinum then may be fixed and relatively immovable, or it may be mobile in its entirety or in part. The displacement of its contents may be slight or marked and may cause few or no symptoms, and again can cause definite symptoms which may seriously affect the course or recovery of the patient.

A combination of these conditions was shown in the chest of a young married woman. The right chest showed a moderate pneumothorax induced in order to get a collapse of the right lung because of a large tuberculous cavity in the right upper lobe. This was not entirely accomplished because of a large adhesion clearly visible. The type of breathing which shows marked complete shift of the mediastinum reveals both diaphragms moving together, the bony chest framework moving together, and, most important, a distinct and separate breathing within the right partially collapsed lung itself. A pneumonolysis was performed and immediate greater degree of collapse of the lung noted, but without complete closure of the cavity. Strangely enough, then, the shift and flutter of the mediastinum was less, but still present, as was more of a pendulum swing. The third study showed the same chest after right phrenic crushing. Marked paradoxical motion of the diaphragm was noted (old side-wheeler motion) but there was no paradoxical breathing. The bony chest moved symmetrically and synchronously on both sides. There was again definitely less shift of the mediastinum and less motion than before, while only a pneumothorax was present. The following summation was made after phrenic operation: (1) marked paradoxical motion of diaphragm; (2) mediastinal shift and flutter greatly reduced; (3) no paradoxical breathing; (4) rise

and fall of bony cage, equal on both sides; (5) on inspiration paralyzed hemidiaphragm appeared to be sucked up.

PARADOXICAL MOTION OF DIAPHRAGM

Of particular interest to us are the studies of paradoxical motion of the diaphragm and of paradoxical breathing. The terms are not synonymous but are unfortunately often confused and interchanged.

Paradoxical motion was described in 1898 by Kienbock.[1] By fluoroscopy he observed that in a unilateral pyopneumothorax the diaphragm with the supernatant fluid ascended during inspiration while the other diaphragm descended normally. This phenomenon he called paradoxical movement. His explanation was that the affected diaphragm was forced up during inspiration by increased intra-abdominal pressure. On the screen this phenomenon was shown by patient Miss C., who had a tuberculous pyopneumothorax on right side. There was normal bilateral rib motion, normal mediastinal movement, an exaggerated left diaphragmatic motion, and a right paradoxical diaphragmatic motion (Kienbock's phenomenon). After second stage thoracoplasty (six ribs) and removal of fluid, the picture changed entirely. There was, of course, considerable collapse, but strangely enough both diaphragms now move synchronously. What has caused this remarkable change? Also, about two weeks later just before her third stage, at which time a moderate amount of fluid and air had reaccumulated, again the diaphragm showed no paradoxical motion.

Paradoxical motion of diaphragm is also caused by loss of nerve conduction through the phrenic nerve by (a) disease (poliomyelitis, etc.); (b) injury, (1) accidental trauma, (2) planned, such as, phrenic crushing, phrenicectomy, phrenic exeresis.

Sauerbruch[2] and later Meyer[5] stated that the cause of paradoxic motion of diaphragm after phrenicectomy is that during inspiration the diaphragm is sucked up through the raising of the thoracic wall, while during expiration, on account of the increase of the intrathoracic pressure, it is pushed down.

Later it was seen, as noted by Schwatt,[4] that the immobility and paradoxical motion of the hemidiaphragm after phrenicoexeresis depend largely on the character of the pulmonary process, especially of the lower lobe, and on the extent and character of pleural adhesions, especially of the basal portion. Also the paralysis depends upon the completeness of the denervation.

Two different cinefluoroscopic views of phrenic crushing effect were shown, one acute and one chronic. The first patient was a man on whom a left phrenic crushing in the neck was done deliberately to paralyze the hemidiaphragm in order to relieve the constant muscular constriction around the neck of a hernia through the paraesophageal space. Cinefluoroscopic studies taken within ten minutes after operation, with the

patient standing, showed a great paralysis of the hemidiaphragm with distinct paradoxical motion, greatly exaggerated, as well as secondary flutterings added to the primary effect—a veritable commotion of contradictory waves of motion and countermotion.

The second patient who had had a phrenic crushing a week before because of a tuberculosis of right lung, moderately advanced, showed a marked contrast to the case just discussed. There was paradoxical motion, but of a settled nature. One hemidiaphragm went up, the other came down in orderly fashion. By this time the chest composed itself to orderly breathing and was more stabilized.

PARADOXICAL BREATHING

In the minds of a good many, paradoxical breathing and paradoxical motion of diaphragm are associated. This most emphatically is not so.

A good working definition of paradoxical breathing is given by Korol.[5] He states that "paradoxical breathing means the deflation of a lung or of a portion of a lung during the phase of expiration."

This process is visible in the human by the contradictory motions of the chest wall or portions of the chest wall, one side of the chest expanding towards the observer while the other is contracting and receding from the observer.

For the respiratory mechanism four factors, anatomically speaking, must be considered: (1) chest wall (semi-rigid); (2) mediastinum; (3) lungs and pleura (free or with adhesions); (4) diaphragm.

Physiologically speaking, paradoxical breathing is constantly present in us; from a portion of one lung to another part and also from one lung to the other.

Again, as shown by Korol, there is normally some paradoxical breathing in the upper portion of human lung because of its anatomical position.

Intrapleural pressure is not the same in different portions of the chest. The pressure and its fluctuations are more marked in the basal portions.[6] In general, the pressure rises in expiration and falls in inspiration. The atmospheric pressure on the chest after a thoracoplasty compresses the operated area more strongly than the rigid portion and so forces some old air into other portions of the same lung through interbronchial communications. This acts as a vicious circle in that dyspnea, already present, is increased by further lack of oxygen.

If these two facts are then correlated, namely, (1) always some paradoxical breathing in upper lung and (2) less protection of lung from atmospheric pressure, we see why there is more apt to be a greater degree of paradoxical breathing after second and third stage thoracoplasties than after the first upper stage.

The length of time and severity of caused symptoms lessens gradually and usually subsides in four to six weeks due to readjustment of contralateral lung and further stabilizing of chest wall by fibrosis and regenera-

tion of bony cage. From what we have observed, the mediastinum plays a minor part in this phenomenon. We do not attempt thoracoplasties now until the mediastinum is fixed or at least so firm that very little fluttering or shifting is present. But still it is in thoracoplasties that paradoxical breathing is most often observed and is always guarded against. Also, under the fluoroscope patients showing paradoxical breathing neither show mediastinal shifting or paradoxical motion of the diaphragm.

In paradoxical breathing the whole bony cage, independent of its contents, has a pivotal swing with its upper portion attached to a fixed point, the vertebral column and lower part swinging from side to side. At the same time the soft parts, mediastinum and diaphragm, have their own rhythm.

Alexander[9] in his recent book also calls attention to the possibility of bronchi partially or wholly blocked with secretions causing paradoxical breathing by impairment of respiration. We have noticed this also in endobronchial lesions in which the patency of the lumen has been diminished or closed. Also it may well be that paralysis of the scalene muscles, or rather loss of their normal functions, besides helping to cause some degree of cervical scoliosis may also increase the tendency towards paradoxical breathing. This would be due to a loss of stability in the upper thorax causing greater inrush of air into the upper lung. This would again demand exaggerated lower breathing with visible paradoxical motion above. Hoover[7] has suggested that a similar condition can be caused by paralysis of the upper intercostal muscles. Certainly this temporary paralysis is always seen following thoracoplasty.

The dyspnea, seen after thoracoplasty when associated with paradoxical breathing, is always apt to be attributed to this cause. But it must be borne in mind that there is also a definite cardiac dyspnea due to cardiac failure. A proper evaluation of both the respiratory and cardiac systems must be made to discover to which the fault belongs.

Haight[8] states that the purposeful temporary preservation of the upper anterior costal stump reduces the incidence of complications that may arise from undue paradoxical motion and that complementary anterior thoracoplasty helps to avoid these. The relative absence of paradoxical motion after the anterior stage is accounted for by the firming of the lateral portion of the decostalized thoracic wall resulting from fibrous tissue in the field of the posterior resection. This, unfortunately, was not the case in a young man who had had three previous partial thoracoplasties in which the whole of the first rib and the larger portions of all the other ribs through the eleventh were resected posteriorly. At the fourth stage the anterior portions of the fifth, sixth, seventh, eighth, and ninth ribs were removed together with the adjacent regenerated bony bridgework. Following this, there was not merely a lagging and inhibition of motion on the operated side, but definitely

paradoxical breathing. In spite of this breathing which has persisted but has not caused undue difficulties, the diaphragms move together, not paradoxically.

The study of patient R. with advanced pulmonary tuberculosis of the right lung was also shown. Before thoracoplasty both diaphragms moved normally. The mediastinum was quiet and showed no shift. The bony cage rose and fell.

After first stage, during the postoperative phase of paradoxical breathing which was never very severe and lasted less than a month, the patient showed (1) a very small shift of mediastinum to normal side on expiration and toward affected side on inspiration, (2) fixation of upper portion of bony cage, (3) slight lagging of diaphragm, but no paradoxical motion (this lagging may be more apparent than real because it is claimed that the central tendon of the diaphragm moves only during very deep inspiration), and (4) a definite but slight paradoxical breathing which at no time caused any dyspnea or discomfort.

SUMMARY

1. In our experiences with thoracoplasties we have seen little or no mediastinal shift in the presence of paradoxical breathing.

2. Paradoxical motion of the hemidiaphragm is also entirely independent of and not necessary to the production of paradoxical breathing.

3. A softer, less resistant or more pliable thoracic covering than is normally present is necessary to cause a manifest paradoxical breathing.

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DISCUSSION

DR. WILLIAM H. STEWART, New York City.—Those who were present at the meeting at Saranac, when I first showed my film, will perhaps appreciate the advancement which I have made in this work during the last year. I stated at that time that the four necessary things in order to obtain more satisfactory work were: (1) A tube which would stand the current for a longer period of time; (2) a screen with more brilliancy than we had; (3) a faster lens; (4) a more sensitive film.

During the last year marked advancement has been made, thanks to the Agfa people, in the development of a film which is four times as fast as the one we used a year ago. You can readily see the advantage of this. We can now extend our exposure to more than 5 seconds and obtain a run of from 16 to 24 frames a second, which is all that is necessary. If we take a 6-second exposure at the normal 16 frames a second we have 96 exposures, which is sufficient to make into a loop and from that loop print as much as is desired.

This film has other advantages. It allows us to be much easier on our tube. We do not need to use as heavy a current. For the average individual we are now using about 90 ma. and around 90 kv.

There is still another advantage to this fast film. It enables us to obtain 24 frames to the second, which is necessary to record sound. We hope not only to show on the screen the heart in action but also to record and amplify in synchronism the cardiac sounds so that students cannot only see the heart beating but at the same time can hear the sounds. The work is progressing slowly but I trust that next year I will be able to show you some further advancement.

DR. POL. N. CORYLLOS,* New York City.—I was extremely interested in the presentation. I think Dr. Stewart and Dr. Thorburn and Dr. Pickhardt are to be commended, especially if they can produce slow motion pictures, which will be extremely interesting.

I do not think the term paradoxical respiration is justified. I think we should replace it by contralateral displacement of the mediastinum. I think the moving pictures have shown very well the cause of the paradoxical respiration. It is the bronchial obstruction. If the bronchus is obstructed on one side the negative pressure becomes much higher in that side of the lung, so the mediastinum is displaced toward the side of the lung were the bronchus is obstructed. Dr. Thorburn and Dr. Pickhardt gave such a demonstration of the above, especially in the case before the last one in which the bronchus was reopened.

In the previous films we saw that the lower lobe did not expand at all and after the opening of the bronchus the lower lobe expanded so that I think it is a matter of replacing the term paradoxical respiration with the term of the etiologic cause, and say it is a pneumolateral displacement of the mediastinum due to the bronchial obstruction.

In another film the speakers showed a forceful expansion of the right upper lobe. There again the explanation is easy. During expiration it was the pendulum movement of the air. During expiration the air was expelled from the freely expanded left lung and it was thrown into the upper lobe of the other side.

DR. JEROME R. HEAD, Chicago, Ill.—Careful observation of patients upon whom phrenicectomy has been performed has shown that in the beginning of inspiration the lower ribs on the affected side move more slowly than those on the normal side. As forced inspiration becomes deeper, they move more rapidly, catching up with the ribs on the opposite side and eventually making a more extensive excursion. The explanation of this asynchrony in the movement of the ribs is as follows: Normally the arch of the diaphragm is such that as the contraction commences it has a lifting effect upon the ribs to which it is attached. As it descends, however, the pull becomes more transverse and tends to oppose the action of the muscles elevating the ribs. Consequently when one-half of the diaphragm is paralyzed the ribs on the normal side move more rapidly at first, but this movement is eventually curtailed by the contraction of the flattened diaphragm.

Recognition of these facts permits one without recourse to fluoroscopy to determine whether or not phrenicectomy has been successful in paralyzing the diaphragm.

*Deceased.

THE DIAGNOSTIC SIGNIFICANCE OF SCOLIOSIS IN INTRATHORACIC DISEASE

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THE various forces that maintain the normal position of the spine are very complex and have to do not only with the ribs, muscles, and ligaments of the thoracic cage, but also with various pressures and stresses that arise within the thorax. An imbalance of any one of these may result in a spinal curvature, the degree of which may be influenced by the type, the location, and the duration of the intrathoracic disease. The diagnostic value of the presence of scoliosis in children has been recognized by radiologists[1] and pediatricians, but it has been given insufficient emphasis and attention.

Having been impressed with the frequency with which the onset of acute empyema is associated with the development of scoliosis, both in children and in adults, we have undertaken a study of the significance of this finding in the differential diagnosis of certain intrathoracic conditions. This has involved the study of 189 cases, including 50 of acute empyema, 39 of pneumonia, 30 of pulmonary infarction, 24 of pleural effusion, 19 of primary, and 11 of secondary intrathoracic tumors, and 16 cases of fractured ribs. The incidence of scoliosis was so much greater in the cases of acute empyema, that its acceptance as one of the cardinal diagnostic signs of this condition seems warranted.

In 50 patients with acute unilateral empyema, 46 (92 per cent) had x-ray evidence of scoliosis before treatment was instituted.

Of the 4 patients who showed no scoliosis, 3 had marked mediastinal shift. In these 3 patients the muscular imbalance, which occurs in an acute intrathoracic pleural infection, was compensated for by a shift of the mediastinum, so that the spine was unaffected (Fig. 1). With the aspiration of pus from the chest, the mediastinum resumed its normal position, and an immediate scoliosis occurred (Fig. 2).

The fourth patient with acute empyema, but without scoliosis, was extremely ill and practically moribund on admission to the hospital, and it may be that the lack of muscle tone caused by the extreme toxicity accounted for the absence of scoliosis.

Of interest in this connection (but not included in the above series) were three patients with bronchiectasis and secondary empyema, none of whom developed scoliosis. All three died, and at autopsy showed iden-

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Read (by title) before the Twenty-First Annual Meeting of The American Association for Thoracic Surgery at Atlanta, Ga., April 4, 5, and 6, 1938.

tical findings—fixation of the mediastinum, the lung adherent to the chest wall, and marked intrapulmonary fibrosis and bilateral bronchiectasis.

The group of patients with fractures of the ribs, though relatively small, showed the next highest incidence of scoliosis, but its occurrence in only 25 per cent of the cases was significantly below that seen in acute empyema. Primary tumors of the lung were associated with scoliosis in 21 per cent of the cases, and metastatic tumors in 9 per cent.

Of great importance, however, from the standpoint of differential diagnosis were the 39 patients with simple pneumonia, the 30 with pulmonary infarcts, and the 24 with simple pleural effusion. Here the incidence of scoliosis was extremely low, being seen in 10 per cent of the pneumonia patients, in 8 per cent of those with pleural effusion, and in only 3 per cent of those with infarction. In these cases the onset of complicating empyema was associated with the development of scoliosis.

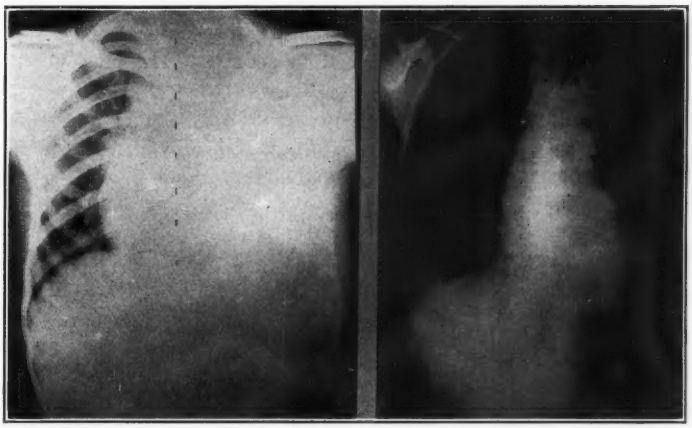

Fig. 1. Fig. 2.

Fig. 1.—Empyema with mediastinal shift. No scoliosis.
Fig. 2.—Same patient as in Fig. 1, following aspiration of pus. No mediastinal shift; scoliosis present.

These findings are of considerable importance in any speculation as to the cause of the scoliosis. For detailed analysis of the mechanical factors involved, the papers of Bisgard,[2] Carey,[3] and Lovett[4] should be consulted, but it would seem that the spinal curvature that accompanies acute empyema is due to the splinting of the intercostal and paravertebral muscles of the corresponding side. It seems, further, that this spasm is an inherent response to infection of the parietal pleura, since scoliosis is infrequent in cases of intrathoracic disease other than acute empyema. That pain alone is of little importance is indicated by the very low incidence of scoliosis in patients with pulmonary infarction. In the latter, pain was an outstanding symptom in 23 out of 30 patients, but in only one did scoliosis develop.

In several cases in which the onset of empyema could be determined with considerable accuracy, it was found that the scoliosis developed at almost exactly the same time. This was seen most strikingly in a patient with typical Type I lobar pneumonia, who was treated with artificial pneumothorax. After induction of the pneumothorax, x-ray at first showed a moderate degree of collapse of the lung with no evidence of pleural fluid and a perfectly straight spine (Fig. 3). On the third day of the treatment the patient became critically ill, and twenty-four hours later, the x-ray revealed a small amount of intrapleural fluid and a slight but definite scoliosis (Fig. 4). Thin exudate was aspirated, which on culture showed pneumococcus Type I.

The degree of scoliosis varies directly with the duration of the empyema and inversely with the age of the patient. Thus, all the patients with acute empyema who were under 15 years of age had marked scoliosis or mediastinal shift. In those over 20 years of age the scoliosis may be slight and easily overlooked, unless special films are taken to demonstrate bone detail. It is important, therefore, to observe the comparative width of the intercostal spaces on the involved side, since this often decreases before true scoliosis has developed or when it is still too slight to be recognized with certainty. This sign is only suggestive, however, for it occurs more often than true scoliosis, not only in cases of acute empyema, but also in patients with pulmonary tumors, fractured ribs, and pneumonia (Table I).

Fig. 3. **Fig. 4.**

Fig. 3.—Pneumonia treated by pneumothorax. No empyema; no scoliosis.
Fig. 4.—Same patient as in Fig. 3, following the development of an empyema and coincidentally a scoliosis.

In the group of patients with acute empyema, there was no evidence that the degree of scoliosis was affected by the type of infection present.

If scoliosis persists after the presumably adequate treatment of an empyema, an undrained pocket of pus should be suspected. Fig. 5 shows the x-ray of a child 10 years of age, who had had postpneumonic empyema treated and supposedly cured four months previously. During the interval, although the child was up and about and never had fever above 37.4°, she failed to gain weight, and x-rays revealed increasing scoliosis and a shadow in the periphery of the left lung, which was interpreted as representing thickened pleura. Repeated aspirations in this area failed to yield any pus. Because of increasing scoliosis, an exploratory thoracotomy was performed. This disclosed a small localized empyema cavity, which contained about 10 c.c. of thick grumous pus, culture of which showed streptococci. Despite the fact that scoliosis had been present for four months, the spine became straight after the pocket was drained (Fig. 6).

TABLE I

	TOTAL NUMBER	SCOLIOSIS		NARROWING OF INTERCOSTAL SPACES	
		NUMBER	PER CENT	NUMBER	PER CENT
Empyema	50	46	92	48	96
Fractured ribs	16	4	25	7	43
Primary tumor	19	4	21	8	42
Pneumonia	39	4	10	13	33
Metastatic tumor	11	1	9	2	18
Pleural effusion	24	2	8	3	12
Pulmonary infarct	30	1	3	4	13

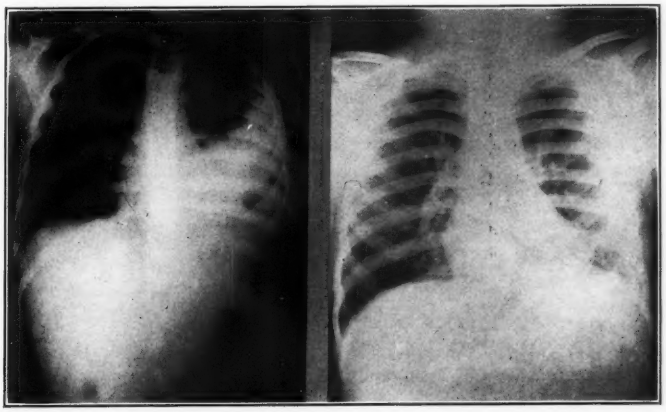

Fig. 5. Fig. 6.

Fig. 5.—Localized empyema with marked scoliosis.
Fig. 6.—Same patient as in Fig. 5. Following drainage of empyema the scoliosis disappeared.

While it is well-recognized that retraction of the chest wall and fixation of the ribs by thickening of the parietal pleura may cause very

refractory or even permanent scoliosis in cases of chronic empyema of long duration, permanent deformity rarely results from scoliosis coincident with acute empyema. No patient in this series has developed permanent scoliosis, although no corrective treatment has been given.

CONCLUSIONS

The high incidence of scoliosis in cases of acute empyema, as compared to other types of intrathoracic disease, warrants its consideration as an important sign in differential diagnosis. In this condition the scoliosis seems definitely to be due to an inflammatory reaction in the parietal pleura, and in degree it varies directly with the duration of the disease, and inversely with the age of the patient. The deviation of the spine disappears promptly after cure of the empyema.

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EXPERIMENTAL OBSERVATIONS ON THE EFFECTS OF CONNECTING BY SUTURE THE LEFT MAIN PULMONARY ARTERY TO THE SYSTEMIC CIRCULATION*

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IN CERTAIN pathologic conditions in man, blood enters the pulmonary circulation directly from systemic vessels. Included among these conditions are congenital cardiac anomalies such at patent ductus arteriosus,[1] acquired fistulas from rupture of an aortic aneurysm into the pulmonary artery,[2, 3] and aberrant pulmonary arteries arising from the aorta or other large arterial trunks.[4, 5] No admixture of arterial and mixed venous blood occurs in the last group nor in the experiments to be described. In all of the above conditions the blood pressure in the systemic vessels supplying the lungs with blood is considerably higher than the pressure usually maintained in the pulmonary circulation. The systemic blood pressure is normally approximately four times the pressure in the pulmonary artery. The present publication deals with the effects of a procedure whereby the left lung receives all of its blood from the left side of the heart.

METHOD

Large mature dogs were anesthetized with ether given by intratracheal catheter. The left pulmonary hilus and aortic arch were exposed through a left intercostal incision (Fig. 1). The left subclavian artery was divided approximately 2.5 cm. distal to its origin from the arch of the aorta. The left pulmonary artery was ligated and divided near its origin. The proximal end of the left subclavian artery was anastomosed to the distal end of the pulmonary artery by the method of Carrel. The lungs were inflated and the wound was closed in layers. There were no significant increases in pulse or respiratory rates following the operation unless empyema supervened. In some of the animals which recovered completely, a small pleural effusion developed which cleared up after thoracentesis.

The mean systemic blood pressure was determined before and at intervals after the operation by puncturing the femoral artery with a needle which was connected to a mercury manometer. After several months had elapsed in which the animals remained in good condition, the oxygen consumption of both the right and left lungs and of the right lung alone was determined. In the latter studies, the bronchus

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to the left lung was occluded temporarily by a ball of cotton. The oxygen content of arterial and of mixed venous blood was determined. The difference in these values divided into the oxygen consumption of the right lung per minute gives one the quantity of blood flowing through the right lung per minute, or in other words, the output of the right side of the heart.

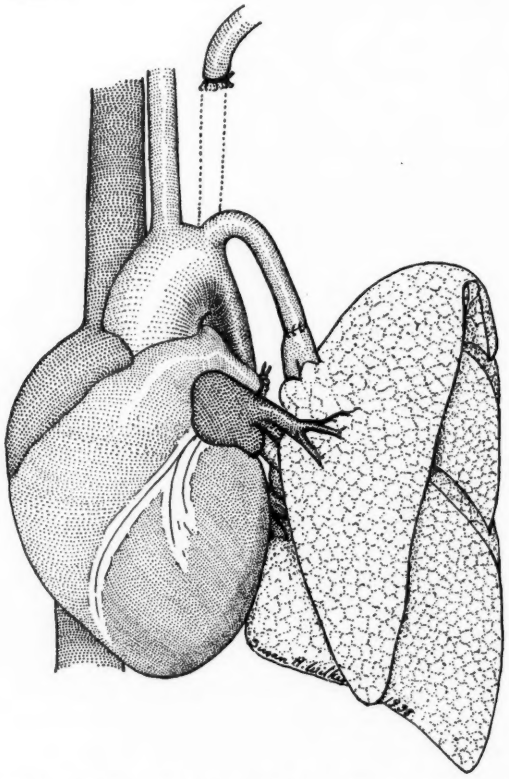

Fig. 1.—Drawing showing method of making anastomosis between left subclavian and left pulmonary artery in dogs.

In order to compare the blood pressures in the pulmonary and systemic vessels, the chest was opened widely under anesthesia with nembutal. Respirations were maintained by supplying air under positive pressure by an intratracheal catheter. The blood pressure was determined by the needle puncture method. Samples of blood for determination of the oxygen content were taken from the right ventricle, right and left pulmonary veins, and the femoral artery. Following these procedures the pulmonary arteries of two of the dogs were injected with Hill's mass[6] after the blood vessels had been washed out with saline solution (Fig. 2).

RESULTS

Five of the twelve dogs which were operated upon survived. Empyema caused the death of six animals and one succumbed to hemorrhage. One of the five dogs which recovered following the operation died on the fortieth day from distemper. Two animals were sacrificed on the ninety-sixth and one hundred sixty-second day, respectively, in order that the pulmonary arteries might be injected with an opaque mass. The remaining two dogs are in excellent condition at the time of writing this paper, 220 and 205 days following operation. The mean systemic pressure was not altered from its preoperative level in any of the animals.

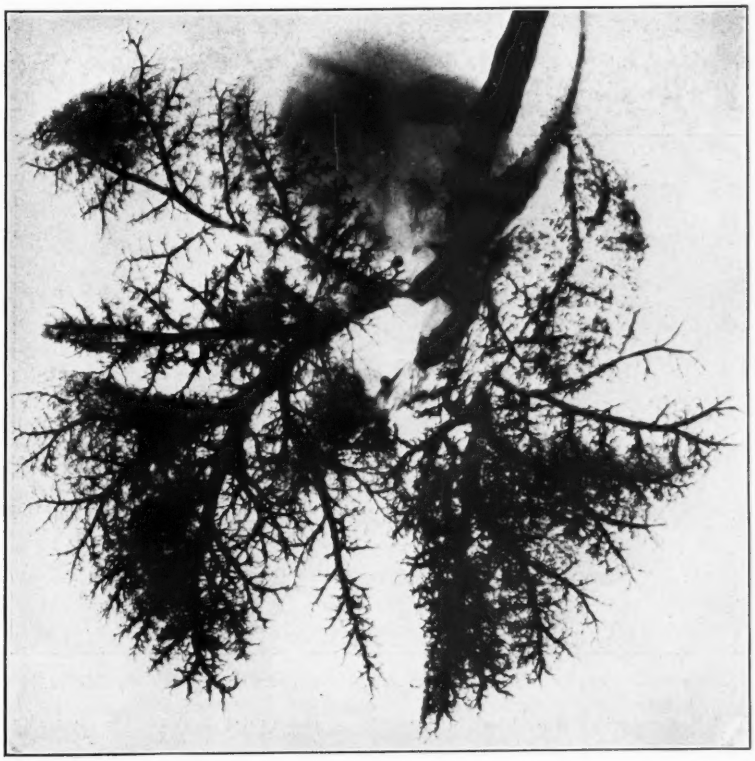

Fig. 2.—Pulmonary arteries injected with Hill's mass. The left lung was injected through the aorta and the right lung through the main pulmonary artery. They were injected simultaneously at the same pressure. Some of the mass escaped from the left upper lobe which was injured in removing it as a result of pleural adhesions at the operative site. The heart is reflected upward.

The blood flow of the right lungs of the two surviving dogs was 1,092 and 1,947 c.c. per minute, the dogs weighing 11.9 and 16.5 kg., respectively. The arterial blood was approximately 99 per cent saturated in both instances. The oxygen consumption of these animals was

59.5 and 76 c.c. per minute. These observations were made on the one hundred fifty-third and one hundred sixty-ninth days following operation. Obstruction of the left main bronchus caused no appreciable alteration in the oxygen consumption. These animals will be observed for a longer period of time.

On inspection of the thoracic contents of two animals under anesthesia, the left lung was quite pink in color compared with the right lung. The left upper lobe was adherent to the chest wall, particularly in the region of the operative incision. Both lungs were aerated, and the respiratory movements appeared normal. Biopsies were taken which showed the histologic structure of both lungs to be normal (Fig. 3). The pulmonary and subclavian arteries at the site of the anastomosis are shown in Fig. 4. An x-ray photograph of the lungs following injection of the arteries with Hill's mass is shown in Fig. 2. There were no striking changes in the architecture of the vessels.

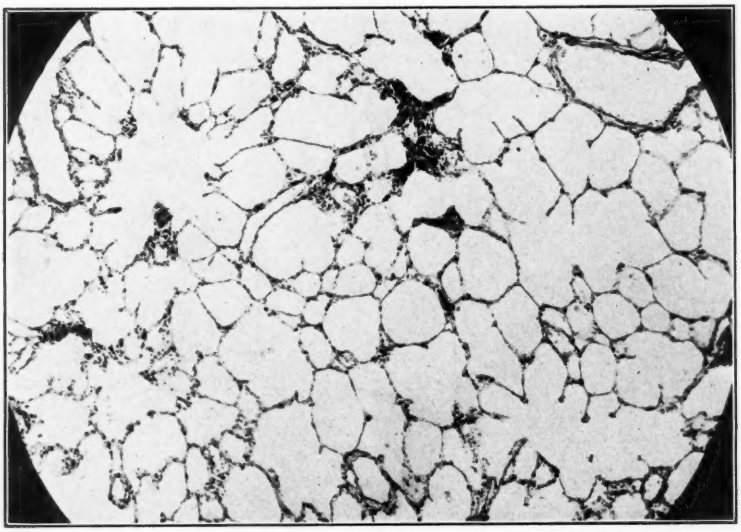

Fig. 3.—Section of left lung 162 days after operation. Hematoxylin and eosin. (x100.)

After opening the chests of two dogs, the blood pressure in the pulmonary and systemic vessels was determined and samples of blood were obtained for analysis of the oxygen content. In both cases the blood pressure in the pulmonary artery a centimeter or two distal to the anastomosis was slightly less than half that in the aorta. The rapid fall in the blood pressure between the aorta and the pulmonary artery is probably due to the low peripheral resistance in the pulmonary circulation. It is likely that the pressure in the peripheral pulmonary vessels was essentially normal. The oxygen content of the blood sam-

ples obtained from the right heart, femoral artery and the left and right pulmonary veins is as follows: right pulmonary vein, 16.60 volumes per cent; right ventricle, 9.60; left pulmonary vein, 19.39; femoral artery, 17.52; oxygen capacity, 19.58. These figures indicate that oxygen was taken up by the left lung. There was 1.87 volumes per cent more oxygen in the blood from the left pulmonary vein than in the sample from the femoral artery corresponding to blood in the left pulmonary artery. However, anoxemia was present, the animal having been under anesthesia for a long period with the chest widely opened. As previously mentioned, observations on dogs without an opening in the chest showed that little, if any, oxygen is absorbed in the left lung.

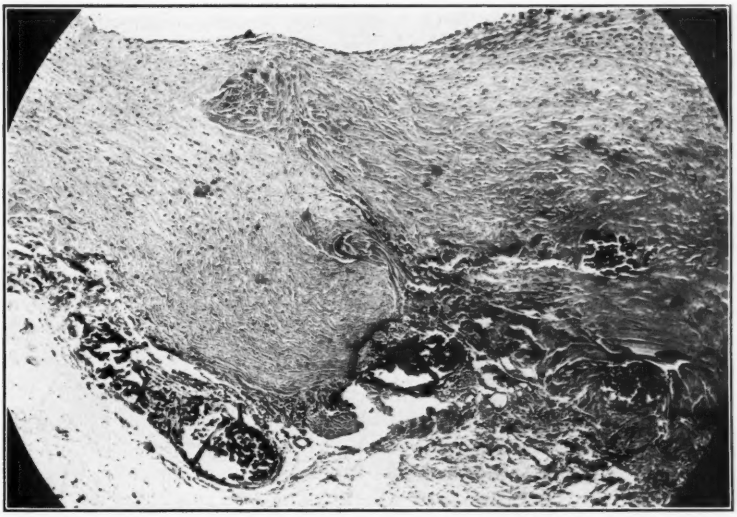

Fig. 4.—Section of site of anastomosis of left pulmonary artery (left) and left subclavian artery. Silk used for the anastomosis is seen in this section. Hematoxylin and eosin. (x100.)

SUMMARY

A method is described by the use of which the left lung receives arterial rather than mixed venous blood. This is accomplished by suturing the proximal end of the divided left subclavian artery to the distal end of the divided left main pulmonary artery. Dogs which have been observed for several months following this procedure appear entirely normal. The left lung was aerated and the respiratory movements were unaltered. The systemic arterial blood pressure was not affected by this operation. The blood pressure in the pulmonary artery only a short distance beyond the anastomosis was less than half of that in the systemic arteries. This was due to the relatively low peripheral resistance in the pulmonary vascular bed. Since only arterial blood

entered the left lung, the quantity of oxygen consumed by this lung was very small. However, when anoxemia was caused, a larger quantity of oxygen was taken up by the incompletely oxygenated arterial blood. The left lung appeared pinker than the right on gross examination during life. Microscopic examination revealed no noteworthy alterations in either the left pulmonary artery or lung.

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FATAL ACCIDENTAL ANOXEMIA RESULTING FROM BRONCHOPLEURAL FISTULA FOLLOWING TOTAL PNEUMONECTOMY

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DURING the last few years, the incidence of bronchopleural fistula following pneumonectomy has been reduced as the result of various refinements and modifications of operative technique. Nevertheless, the complication still occurs in a substantial number of cases especially when the condition for which operation is performed is associated with infection.

When contamination occurs or gross infection is present within the operative field at the time of operation, the residual intrapleural dead space usually is drained by the closed method. Under such circumstances if the bronchial stump subsequently opens and frank infection of the pleura results, a bronchopleurocutaneous communication is formed. If for good reason the intrapleural dead space is not drained at the time of operation and the bronchial stump subsequently opens and results in infection of the pleura, secondary pleural drainage usually is required. Thus under any circumstances, following pneumonectomy the bronchopleural fistula associated with pleural infection practically always communicates with the outside through a thoracic opening made for the purpose of drainage.

Such bronchopleural communications may close spontaneously, essentially by one of two mechanisms. The first is by the growth of peribronchial granulation tissue which gradually bridges the bronchial defect. The second is by the progressive obliteration of the intrapleural dead space which results in the apposition of the open bronchus to the chest wall. In cases in which the fistula closes before the intrapleural dead space has become obliterated, the first mechanism alone usually is operative. In cases in which pleural obliteration is necessary for final closure of the fistula, although growth of granulation tissue takes place about the bronchial opening it obviously is inadequate. During the period in which the intrapleural dead space is being obliterated, a process which may require several months, the bronchopleural fistula is in more or less free communication with the outside through the thoracic wound except at such times as the latter is occluded by packings or dressings. When the bronchopleural fistula or the external opening or both are small, the existence of the external opening per se as a rule does not produce significant respiratory em-

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barrassment provided the mediastinum is well fixed. If on the other hand the bronchopleural communication is large, considerable respiratory embarrassment may occur in the presence of an external opening of moderate size even after the mediastinum has become fixed. This is illustrated by the case about to be presented.

In brief, following pneumonectomy for carcinoma, and drainage of a secondary pleural infection, the patient was left with a large bronchopleural fistula and an open pneumothorax. During dressings, when the packings were removed from the empyema cavity and the bronchopleural fistula thus was in communication with the outside, the patient became dyspneic and cyanotic and was unable to speak in a voice above a whisper. As soon as the external opening was occluded with a packing or the hand, the symptoms disappeared. This syndrome persisted for the entire period of six months (during which the patient lived following operation) and appeared to be related to his death.

A. S., a male, aged 63 years, and a laundry worker by occupation, was admitted to the Medical Service of The Mount Sinai Hospital on July 8, 1937. The important features of the history were as follows: About one year previously, paroxysmal cough began. This occasionally was productive of yellowish sputum. Cough was worse at night and usually was relieved by the orthopneic position. Shortly after onset, intermittent pain in the lower part of the right chest and marked dyspnea on exertion were noted. During the ten weeks preceding admission, there had been a loss in weight of 14 pounds.

Three days prior to admission the patient developed increased cough, temperature of 104° F., and felt cold and weak. At the same time he became quite dyspneic, and suffered a recrudescence of pain in the chest.

Except for pneumonia thirty years previously, the past thoracic history was negative. The patient's wife, however, had suffered from pulmonary tuberculosis for the past ten years.

On physical examination, the following findings of significance were noted: Temperature 103.6° F.; pulse 92; respirations 26. The patient was somewhat emaciated, his voice was hoarse, and he was coughing frequently. There was no expectoration. Pulmonary resonance over the left apex and both bases was diminished. There was no clubbing of the fingers nor cyanosis. The remainder of the physical examination was irrelevant.

Laboratory findings were as follows: hemoglobin, 75 per cent; W.B.C., 16,500; polymorphonuclear leucocytes, 74 per cent; lymphocytes, 22 per cent; mononuclears, 4 per cent; sedimentation rate 12 minutes. X-ray examination of the chest revealed mottled consolidation of the right lower lobe, indicative of a pneumonic process (Fig. 1).

Although the episode which brought the patient to the hospital was of only three days' duration, he had suffered chronic pulmonary symptoms for about a year and had been in contact with a known case of tuberculosis for ten years. These facts aroused the suspicion that the acute illness might be tuberculous in nature and accordingly the sputum was examined for tubercle bacilli. This proved negative and smears from the larynx then were examined. When these too proved negative, bronchoscopy was performed (four days after admission) in order to obtain direct smears from the bronchi for further examination. The report of the bronchoscopist follows:

"The right lower lobe bronchus is markedly narrowed by an intrabronchial mass and submucous infiltration. The mass is grayish, irregular, and hard; and arises from the postero-lateral wall distal to the orifice of the middle lobe bronchus. Thick, creamy, odorless pus emanates from the stenosed bronchus and keeps reappearing when the latter is dilated. Impression: Carcinoma of the right lower lobe bronchus with secondary infection." A specimen was removed for pathologic examination.

Following bronchoscopy and dilatation of the stenotic bronchus, the temperature began to fall and four days later was normal. In the meantime the cough had diminished considerably. A few days later the pathologic specimen was reported as follows: "Squamous cell carcinoma with hornification."

A review of the x-ray films failed to reveal evidence of mediastinal lymph node involvement, and a second physical examination failed to disclose the presence of metastases elsewhere. Accordingly, operation was advised. Pneumothorax therapy was begun in preparation for operation, but because of the presence of extensive intrapleural adhesions it was impossible to produce an appreciable degree of pulmonary collapse. X-ray examination thirteen days after admission revealed the infiltration within the right lower lobe to have diminished markedly. Only a very small, localized area of pneumothorax was present immediately above the costophrenic sinus (Fig. 2).

Operation was performed on July 27, 1937, under gas-oxygen and avertin anesthesia. An anterior thoracic approach was made in the fourth intercostal space. The fourth and fifth costal cartilages were divided through a vertical extension of the incision, and the pleura opened. In the exposed operative field, the lung was broadly adherent to the parietal pleura. A plane of cleavage between lung and pleura was found, and the lower lobe was dissected free in all directions. As the dissection was continued, the lobe was found firmly agglutinated not only to the thoracic parietes, but to the mediastinum, pericardium, and diaphragm as well. With considerable difficulty, the middle and lower lobes were freed from the interior of the thorax. At several points the parietal pleura was so adherent that it had to be detached with the lung, from the endothoracic fascia. The original plan was to remove only the middle and lower lobes, but these were so densely adherent to the upper lobe, that a plane of cleavage between them and the upper lobe could not be found. Several attempts to enter a plane of cleavage between the lobes resulted in brisk bleeding. Accordingly, it was decided to proceed with pneumonectomy rather than to risk the danger of serious hemorrhage. The upper lobe, which also was densely adherent to the interior of the thorax on all aspects, was freed, therefore, and the lung mobilized. Palpation of the mediastinum failed to reveal any glands which appeared to be grossly involved by neoplasm. At the hilus, several small anthracotic glands which appeared free of disease were noted. After ligation and division of the inferior pulmonary ligament, a tourniquet was placed about the hilus and the lung ablated. The pulmonary vessels were ligated individually, and the bronchus then was transfixed and ligated in two sections with chromic catgut. The peribronchial tissues at the hilus were approximated over the bronchial stump and vessels, with fine interrupted chromic sutures. The phrenic nerve was isolated and crushed. A small caliber rubber tube was introduced, through a stab wound in the tenth intercostal space, for the purpose of closed drainage. The fourth and fifth ribs were approximated with heavy pericostal sutures, and the overlying musculature closed in layers with interrupted sutures. The skin was closed with silk.

The following is a summary of the pathologic report upon the excised specimen: Squamous cell carcinoma of the right lower lobe bronchus with hornification. Chronic bronchitis and chronic lobular pneumonia of the right lower lobe. Fibrous pleural adhesions. No lymph node metastases.

The patient's reaction following operation, aside from irregular temperature for the first four days, was satisfactory. During this period, the pulse rate ranged

around 84 and respirations 20. Dyspnea and cyanosis were slight. Drainage, which at first was bloody and profuse, decreased rapidly. On the eighth postoperative day the patient was x-rayed. The heart and mediastinal structures were found shifted well toward the side of operation, the diaphragm was well elevated as the result of phrenic interruption, and a moderate amount of fluid was present in the right chest.

On the seventeenth postoperative day, the discharge through the drainage tube became purulent. Simultaneously the temperature rose and became irregular, and bubbles of air were noted escaping from the underwater drainage tube. The evidence that the bronchial stump had opened widely now was clear, and closed drainage was discontinued and a short open-end tube was introduced. Because of continuation of irregular fever, a short rib resection was performed under local anesthesia a few days later (twenty-one days after pneumonectomy). At operation, a small amount of slough at the hilus and an open bronchus which measured about 1 cm. in diameter were noted. The interior of the thoracic cavity was fairly clean except for the presence of a single large mass of fibrin. The latter was removed and the cavity lightly packed with gauze.

From then on, the patient made steady progress and began to gain in weight and strength. The empyema cavity, which became progressively smaller, was kept lightly packed with gauze. During dressings, when all packings were removed from the cavity, the bronchial fistula could be seen readily and hissed noisily. The latter was determined to be due to the escape of air as the patient inspired. Almost simultaneously with this escape of air the patient would become dyspneic and cyanotic and would have difficulty in speaking in a voice much above a whisper. For these reasons the dressings always were performed with dispatch, for as soon as the external thoracic opening was occluded the symptoms would disappear. In order to maintain effective occlusion, when the external wound became small it was stoppered with a rubber plug the caliber of which was gradually reduced as the opening became narrower.

Because of unfavorable home conditions, the patient was kept in the roof ward for five and one-half months. During this period the bronchopleural fistula remained widely patent in spite of attempts at closure by means of repeated topical application of 40 per cent silver nitrate according to the method of Adams and Livingstone. Moderate mucoid discharge from the open bronchus continued, but in spite of low-grade fever due to mild residual infection of the intrapleural dead space, the patient was up and about, felt well, and gained weight. Dyspnea, cyanosis, and difficulty in phonation continued to recur when the plug was removed from the external thoracic opening, only to disappear as soon as the plug was replaced. Although the bronchial opening, which was flush with the mesial surface of the intrapleural dead space, could no longer be visualized through the thoracic opening, it continued to blow freely when the latter was open. The dead space however diminished progressively in all diameters, and at the time of the patient's transfer to another institution at the end of five and one-half months it had become converted into a narrow cleft (Fig. 3). Because of this it seemed warranted to assume that the cavity would become obliterated and the fistula would close spontaneously within a reasonable period of time. The advisability of removing the plug and permitting the external sinus to heal was considered, but because of the continuation of mucoid discharge from the fistula it was decided to keep the plug in place in order to permit the evacuation of the contents of the cavity daily.

One afternoon, about two weeks after arriving at the second institution, the patient gradually became dyspneic and cyanotic and had difficulty in speaking. Recognizing these symptoms, he requested that his wound be inspected. Due to unavoidable delay this was not done until about two and one-half hours had elapsed, but by that time he was moribund and died shortly thereafter.

At post-mortem examination, the following significant findings were noted: The right pleural cavity contained no pulmonary tissue and measured about 10 by 5 by 5 cm. It was lined by greatly thickened pleura which ranged from 0.5 to 3 cm. in thickness in various areas. An external sinus in the posterior axillary line entered directly through the thickened pleura into the pleural space. The internal opening of this sinus measured 1 cm. in diameter. High up in the pleural space a large smooth, circular opening which measured 1 cm. in diameter was found. This entered directly into the upper lobe bronchus. The remainder of the hilar structures were completely covered by thick pleura. Several large anthracotic, fibrotic lymph nodes were present in the hilar area but there was no evidence of tumor tissue present.

Fig. 1. Fig. 2.

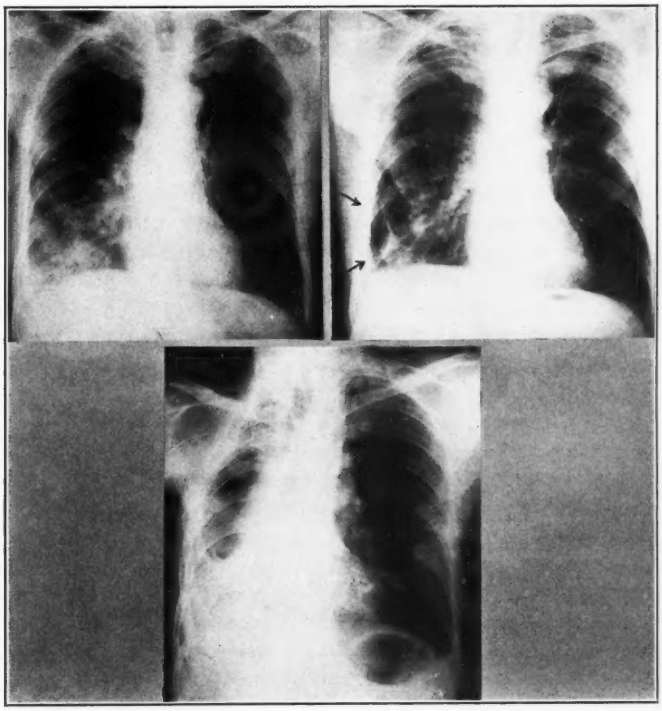

Fig. 3.

Fig. 1.—Admission Film. Note consolidation involving the right lower lobe. Remainder of pulmonary fields normal.

Fig. 2.—Thirteen days later. Note subsidence of pneumonic process following bronchoscopic removal of specimen and dilatation of right lower lobe bronchus. Unsuccessful preoperative pneumothorax due to extensive intrapleural adhesions. (Air pocket indicated by arrows.)

Fig. 3.—Five and one-half months postoperative. Note marked reduction in size of intrapleural dead space as the result of mediastinal shift, elevation of diaphragm, and contraction of the thoracic cage.

Thickened pleura was firmly adherent to the pericardium. The heart was shifted so that the edge of the right auricle was 4 cm. beyond the right sternal border, and firmly fixed in that position. The right diaphragm was elevated to the upper border of the fourth rib. The left lung was voluminous and filled the entire left pleural

space. It was crepitant and emphysematous throughout. There was some mucopurulent secretion in the lower lobe. The right ventricle was dilated and hypertrophied.

Microscopic examination of the heart, lungs, cervical and mediastinal lymph nodes, bronchial stump, the right pleura, liver, spleen, pancreas, suprarenals, kidneys, prostate gland, testes, thyroid, parathyroid, and ribs about the site of the thoracic opening, failed to reveal the presence of carcinomatous involvement. Permission to remove the brain and spinal cord was not granted.

In the absence of any demonstrable anatomic cause of death, the history of the mode of exitus appeared to warrant the assumption that the packing, which had been substituted for the rubber plug, had become accidentally loosened with the result that the patient became increasingly dyspneic and anoxemic and finally expired. Although the brain and spinal cord were not examined, the symptoms preceding death did not suggest the presence of an intracranial complication as the lethal factor.

DISCUSSION

A consideration of the mechanism of the dyspnea, cyanosis, and difficulty in phonation which the patient suffered, when the bronchopleural fistula was in communication with the outside, is of interest. The difficulty in phonation will be dealt with first. During speech the vocal cords are adducted in varying degree and are actuated by an expiratory flow of air from the trachea. This adduction of the cords increases the resistance to the outflowing current of air, so that in order to force the air past the cords and vibrate them the intratracheal air pressure must be increased voluntarily by expiratory effort. An adequate increase in intratracheal pressure is impossible if free leakage of air occurs at any point in the tracheo-broncho-pulmonary system. Thus as our patient attempted to speak while the thoracic wound was open, air leaked freely from the open bronchial stump to the outside and the intratracheal air pressure could not be raised sufficiently to actuate the vocal cords normally. The result was a peculiar type of harsh whisper. When the thoracic opening was occluded, the system immediately was converted into one of the closed type; and although there was some leakage from the open bronchus into the intrapleural dead space, the pressure within that space was quickly built up to become essentially equal to the pressure within the trachea and the bronchi of the intact lung. As the result of the restoration of normal intratracheal pressure, the vocal cords then could be vibrated normally.

A satisfactory explanation of the cyanosis and dyspnea throughout the five and one-half months' period of personal observation is more difficult. One fact, however, is of importance, namely, that when the open pneumothorax was converted into a closed one the symptoms were alleviated. It may be assumed that when thoracotomy and rib resection were performed twenty-one days after pneumonectomy, the mediastinum although obviously stiffened was not firmly fixed. Thus on inspiration some retraction of the mediastinum toward the normal

side could be noted. At the same time, air hissed freely into the empyema cavity from the open bronchus. The flow of air from the bronchus on inspiration could have been due only to a tendency toward increased negativity within the empty right chest. Such increased negativity was the result of inspiratory retraction of the mediastinum and elevation and rotation of the ribs. As the capacity of the right chest thus was increased, air rushed in from two sources, namely, the open bronchopleural fistula and the external thoracic opening. It appears warranted to conclude that as the result of the free escape of air from the open bronchus, the quantity of air entering the intact left lung on inspiration was insufficient to carry on adequate oxygenation. The resulting partial anoxemia, therefore, was responsible for the cyanosis and dyspnea. When the external thoracic opening was occluded, although the same tendency toward increased negativity remained within the empty right chest on inspiration, the pressure of the air which collected within the cavity tended to equal the pressure of the air in the trachea and the bronchopulmonary tree of the intact left lung because the system was now of the closed variety. As a result of the tendency toward stabilization of these pressures, the left lung could inflate sufficiently to permit adequate oxygenation. Removal of the occluding plug from the external thoracic opening, by upsetting this equilibrium would result in prompt recurrence of symptoms.

With the passage of time the external thoracic opening, which was situated in the lower part of the chest, narrowed down to a caliber of 1 cm. The mediastinum became firmly fixed. The bronchopleural fistula which was situated high in the chest no longer could be seen through the small thoracic wound but its diameter did not diminish appreciably (as demonstrated at post-mortem examination). Because of the narrowness of the external wound, the direction of the flow of air at the mouth of the bronchopleural fistula in relation to the phase of respiration no longer could be tested. At this stage, the intrapleural dead space was of small size and was lined by thick, fibrous tissue on all aspects. The mediastinum now was firmly fixed. In the presence of a shrunken cavity, a fixed mediastinum, and a thoracic cage immobilized by dense scar tissue which lined its interior, it appeared warranted to assume that there could be very little if any increase in negativity within the cavity on inspiration. Accordingly, at this stage of observation it was necessary to invoke some other theory of the continuation of symptoms. Although there are several possibilities, none of the explanations are entirely satisfactory and the author still is in doubt as to the exact mechanism involved. It should perhaps be emphasized that even many months after operation, relief of symptoms still could be obtained by occluding the external thoracic opening.

In addition to the unusual complication to which reference already has been made, the case presents a number of other interesting features. From the clinical standpoint, the patient's presenting symptoms were those of an acute pneumonitis engrafted upon a one-year history of cough and expectoration and a ten weeks' history of loss of weight and thoracic pain. Because of the history of pulmonary tuberculosis in the patient's wife, and the presence of physical signs and roentgenographic findings which were not inconsistent with the diagnosis of acute pulmonary tuberculosis the latter was considered most strongly. When the sputum and smears from the larynx proved negative for tubercle bacilli, bronchoscopy was performed and a carcinoma obstructing the right lower lobe bronchus was disclosed. The acute pneumonitis, which involved the bronchopulmonary segments distal to the site of obstruction, subsided promptly when the obstruction was relieved by removing a portion of the growth and dilating the bronchial lumen at that point.

From the surgical standpoint, several features are worthy of comment: first, the unusually dense and extensive adhesions which united all the lobes not only to one another, but also to the entire interior of the hemithorax. (It is to be emphasized that the carcinoma involved only the right lower lobe bronchus.) These adhesions not only prevented the instituting of pneumothorax preoperatively, but also made it necessary to perform pneumonectomy in order to avoid the hemorrhage which would have occurred if one had persisted in the attempt to remove only the middle and lower lobes. The second feature was the absence of grossly involved hilar or mediastinal lymph nodes at operation. This finding was confirmed by examination of the excised lung which failed to disclose the presence of involved glands (and also indicated that the tumor had been widely removed).

The features of the post-mortem examination were the failure to find evidence of local recurrence or distant metastases, and the absence of an anatomic cause of death.

Aside from adding further evidence of the operability of certain cases of carcinoma of the major bronchi, the case illustrates one of the dangers of persistent postpneumonectomy bronchopleural fistula of large size in association with open pneumothorax. It should also serve to emphasize the necessity of closely supervising such cases until the open bronchus has closed completely.

VENOUS PRESSURE IN PULMONARY TUBERCULOSIS

The Effect of Collapse Therapy and Other Complications

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THE measurement of venous pressure was first accomplished by the pathologist, Stephen Hales, in 1733. It was not until the present century that very much was done in the clinical use of venous pressure determinations. The failing right side of the heart was found to give characteristic findings. Other diseased processes were found to affect venous pressure little, if at all. Not very much work has been done in the determination of venous pressure in tuberculosis, although in the literature on tuberculosis there are references to isolated cases.

In 1909, Plumier[1] conducted some experiments on dogs. He found that the injection into the pleural cavity of a liter and a half of fluid over a period of ten minutes caused a considerable increase in the respiratory rate, an increase of the carotid venous pressure, a stationary venous pressure in the jugular vein, and a moderate increase in the inferior vena cava. Five minutes later the animal received close to 3 liters of fluid into the pleura, which caused a further increase in respiratory rate and a further increase in carotid venous pressure, an increase in the inferior vena cava, and in the external jugular vein. He found that if fluid was injected in sufficient quantities to lead to the death of the animal, there was a marked and rapid rise in venous pressure very comparable to that obtained in suffocation.

The monograph of Villaret, Saint Girons, Justin-Besançon,[2] published in 1930, applies in a very limited way to our present interest in tuberculosis and pneumothorax. In studying the effect of respiration on venous pressure they discovered (1) that during inspiration, even forced inspiration, there was no modification of the venous pressure; (2) that in expiration, slight or forced, no modification of pressure was noticed, contrary to Wertheimer, who found it was lowered; (3) that during voluntary hyperpnea the pressure rose markedly; (4) that during the Valsalva experiment or prolonged expiration pressures may rise to 200, 250, 300, or 400 mm.; (5) that during Müller's experiment, which consists of making an inspiratory effort in the expiratory position, with closed glottis, the inverse result was obtained, pressure falling 40 to 50 mm.; (6) that a simple nonspasmodic cough raises the venous pressure 20 to 30 mm. During pleural effusions, as Cordier has shown, only collections in the great pleural cavity on the right seem to raise the venous pressure. He believed that this was the result of a direct pressure on the superior vena cava. Villaret and others believe

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that this action is not limited to the superior vena cava, but can occur when pressure is put on the thin-walled resilient portion of the auricles, in effect, whenever pressure is applied in the cardiovascular zone corresponding biologically to the venous sinus.

In 1927 Villaret and Martiny[3] described what they believed to be a primary hypertension of the lesser circulation in patients having pulmonary tuberculosis during the prodromal symptoms of hemoptysis. Their interest was again aroused by the radioscopic work of Laubry, Chaperon, and Thomas.[4] These latter authors believed that they could differentiate radioscopically between hypertension of the pulmonary artery and hypertension of the pulmonary vein. Villaret and Martiny, using this radioscopic technique, found that during hypertension of the pulmonary artery the venous pressure was always high, that during hypertension of the pulmonary vein the venous pressure was never high, and that in the association of both of these syndromes, both physical signs and high venous pressure were present.

Kroetz,[5] in 1922, was apparently the first to study the venous pressure of patients before and after pneumothorax. He found that of six with pneumothorax, the four with complete collapse had a simultaneous rise in venous pressure as the pleural pressure became more positive. In the one case of this group which he describes in detail, the pleural pressure rose from –5, –2.5 to –2.5, 0 while the venous pressure rose from 71 to 95 mm. after the giving of 400 c.c. of air. In this particular case the pneumothorax and venous pressure were both of the right side. He states that in the three other cases the results were similar, whether the venous pressure was taken on the right or left side. In two cases of partial pneumothorax, in one of which the upper lobe was completely adherent to the chest wall, the venous pressure fell as the pleural pressure became positive. In the example given, the pleural pressure rose from –3, –1 to 0, +1½ while the venous pressure fell from 75 to 45 mm. Regardless of whether the pneumothorax was complete or partial, multiplication of the venous pressure by the average pleural pressure gave essentially the same result or coefficient.

In 1925 Villaret and Martiny[6] studied the venous pressure in 200 cases of tuberculosis, the details of which were published in Martiny's thesis. They found venous pressure increases in miliary tuberculosis, bilateral progressive ulcerocaseous tuberculosis, pleural effusions, particularly on the right side, and in two cases of hemoptysis; in these latter cases, the hemoptysis was preceded by prodromal symptoms and the venous pressure increased from 40 to 100 mm. They believed that whatever the pathologic cause of the hemoptysis, it was preceded by a vascular engorgement of the lesser circulation.

In administration of therapeutic pneumothorax they found that the first insufflation had little influence on the venous pressure. From the second or third insufflation the venous pressure rose from 10 to 40 mm.

At this stage there was no apparent modification of the cardiomediastinum from the clinical or x-ray evidence. In the following insufflations the venous pressure rose less, the heart accommodated, and still later the venous pressure sometimes rose 40 to 80 mm., sometimes very sharply. In these cases there was usually a marked displacement of the heart. The influence of this displacement was more intense and earlier when produced from right to left. Sometimes a late elevation was seen, that is, at the sixteenth or eighteenth refill and preceded the clinical symptoms of intolerance.

Grellety-Bosviel[7] also found that as a rule the establishment of pneumothorax produces no modifications of venous or arterial pressure. In 210 patients with unilateral pneumothorax established from some months to many years, there was no modification of venous pressure in 85 per cent. In 15 per cent there was a temporary or prolonged elevation. When elevation occurred, he considered it due, not to the difficulty of penetration of blood into the collapsed lung, but to the compressions of the cardiac cavities and their vessels by the following factors: too much air, pleurisy, and pleuropulmonary sclerosis with adhesions of the mediastinum. Of twelve patients having dyspnea, palpitation, or bouts of fever after refills, ten had an elevation of venous pressure ranging from 150 to 180 mm. This hypertension persisted a day or two, occurred in the presence of negative pleural pressures, and became normal with a reduction in the amount of air given. He found that an astonishing amount of mediastinal displacement can be tolerated without a rise in venous pressure due to the flexibility of the tissue, although where pleurisy was a complicating factor, pressures of 170 to 190 mm. were not unusual. He found the venous pressure unchanged in four patients with bilateral pneumothorax. In eight patients with spontaneous pleural fistula, interestingly enough, he found that the abrupt, even total collapse of even the right lung did not modify the venous pressure. Only when massive effusions formed, or in the presence of a valve pneumothorax, did these patients develop high venous pressure.

Wartman,[8] in a study of thirteen patients with tuberculosis, concluded that the venous pressure was not influenced by the disease.

Villaret, Saint Girons, and Grellety-Bosviel[9] concluded from their studies that the venous pressure in pulmonary tuberculosis is usually low, in which case there is no restriction of the right heart (acute forms and caseous forms sometimes with terminal cyanosis), while in other cases where the tendency to fibrosis tends to obstruction of the pulmonary circulation, with or without retraction of the right heart, it is elevated.

Cordier[10] found that in a left pneumothorax rapidly induced for the control of hemorrhage (with an initial injection of 700 c.c. repeated the next day with a 1000 c.c. injection) dyspnea occurred when the venous pressure rose from 130 to 260 mm. In an induced right pneumothorax,

with an injection of 300 c.c. of air, the venous pressure rose from 130 to 200 mm., symptoms of intolerance developed rapidly, the patient died, and the autopsy showed the heart affected by an old pleurisy on the left. In a left pneumothorax, undertaken for social reasons, venous pressure rose rapidly from 140 to 160 mm. after the injection of 3100 c.c. of air, in three sittings; tolerance was perfect without any cardiac symptoms, regardless of mediastinal displacement. He concludes that in pneumothorax venous pressure is a valuable indication of circulatory obstruction not always detected by clinical observation or by x-ray study; in the rapid induction of pneumothorax venous pressure should be taken before and after the first injection and during the second; venous pressure can predict that the collapse should be interrupted even when auscultation, the x-ray, and the respiratory rhythm sometimes fail to forecast disaster.

Villaret and Desoille[11] concluded from their studies in therapeutic pneumothorax that a late rise in venous pressure, say at the sixteenth or eighteenth refill, constituted a true sign of alarm which preceded the symptoms of intolerance.

Overholt and Pilcher[12] studied the venous pressure in both arms of thirty-five thoracoplasty patients with normal preoperative venous pressure. In eleven of these there was a unilateral elevation following operation, usually the upper stage. When the first stage was limited to from three to five upper ribs, there was little likelihood of disturbing the previously normal venous pressure. When a second stage followed within two weeks of the first stage, an elevated venous pressure on the operated side could be expected. The resection of more than four ribs frequently disturbed the venous pressure on the operated side. They were impressed by the relationship between an elevated venous pressure postoperatively and what was considered poor tolerance to the amount of collapse provided. An elevated venous pressure seemed to indicate an excessive degree of collapse. They considered the measurement of venous pressure in thoracoplasty cases of value for two reasons: First, it aided in establishing the extent of rib removal which would insure a wide margin of safety during convalescence; second, it was one of the factors which convinced them that a second operation should not be done so soon after the first, that the effect of the two equaled that brought about by a too extensive single operation. It is very interesting that the significant alterations were found on one side only or predominantly on one side and, therefore, were considered to be of mechanical origin, as distinguished from the generalized elevations of cardiac origin. Nevertheless, even though the right heart was found intact by the most sensitive method we now possess of evaluating its function, many of these patients showed marked signs and symptoms of circulatory embarrassment following operation.

WHAT IS NORMAL VENOUS PRESSURE?

Obviously, whether or not venous pressure is elevated depends on what one accepts as normal. Unfortunately, there is considerable difference of opinion on this subject. Bedford and Wright[13] tabulated the various opinions in 1924 and we have brought this table up to date.

TABLE I

OBSERVER	METHOD	VENOUS PRESSURE
Frank and Ray	Indirect	10-60
Eyster and Hooker	Indirect	50-100
Hooker	Indirect	Upper limit -200
Moritz and Tabora	Direct	40-80
Overholt and Pilcher	Direct	Upper limit -120
Castellotti	Direct	40-70
Wartman	Direct	60-120
Corradi	Direct	80-130
Villaret and Others	Claude's man	120-130
Gazzotti	Claude's man	130-140
Bedford and Wright	Claude's man	50-150
Eyster	Not stated	Upper limit -110

In the light of these figures, then, it appears that 120 mm. of water is regarded as the average upper limit of normal venous pressure by most authors and this is the figure which we have adopted for this study. However, even though we have yielded to the greater experience of many authors, it is our opinion that normal venous pressure may, under certain conditions, be much higher than this and that 150 mm. might well be taken as the dividing line. In fact, we have seen at least two instances of pressures close to 200 mm. which might well be considered normal. Deviation from basal conditions and emotional factors particularly raise the venous pressure. We are inclined to think of venous pressure determinations as being somewhat like the basal metabolic rate in that a disturbance, however slight, of the basal conditions will raise both of them.

CASES INCLUDED IN THE PRESENT STUDY

A deliberate effort was made to select cases of tuberculosis, with or without complications, which on the basis of what we already know would be likely to give high venous pressures. Certain cardiac and pulmonary complications were included in the study, but our particular interest was in pneumothorax and thoracoplasty cases. All patients in the present study had their venous pressure determined after they had been in a reclining position for at least fifteen minutes and some for many months. The direct method was used with homemade citrated water manometers and 16 gauge needles. When possible, pressures were taken in both arms simultaneously. In the series of twenty-one cases with reports of simultaneous venous, arterial, and pleural pressures, and the volume of air injected, the same method was used for

determining venous pressure and an ordinary sphygmomanometer was used by another observer on the opposite arm. A third physician gave the pneumothorax and readings were dictated, at intervals of a few seconds, to a secretary.

RESULTS OF THE PRESENT STUDY

Arterial Pressure.—The effects of pneumothorax on arterial pressure were reported by Steidl and Heise[14] and the essential fact of a drop in pressure agreed with the work of Grant.[15] In the present study the arterial pressure was found to follow the general pattern described by Steidl and Heise. The fluctuations in degree of this pattern were not so marked, however, in the present study, possibly due to the smaller number of cases and possibly due to the more rigid basal conditions maintained in the present study.

TABLE II

VENOUS PRESSURE IN CARDIAC COMPLICATIONS OF PULMONARY TUBERCULOSIS

COMPLICATION	ARTERIAL PRESSURE	VENOUS PRESSURE	
		RIGHT	LEFT
Essential hypertension	210/130	136	139
Essential hypertension	170/110		135
Hypertensive heart disease	200/120	78	78
Cardiovascular-renal disease	230/130	90	95
Toxic myocarditis		100	
Toxic myocarditis		61	56
Mediastinal and cardiac retraction to left with dyspnea		68	80
Paroxysmal auricular flutter (between attacks)	110/88	96	

Cardiac Complications.—In the cardiac complications, most of which consisted of severe hypertension, it will be observed that only two patients had a venous pressure above 120 mm. Since it is recognized that hypertension itself will sometimes give borderline readings, it is difficult to ascribe these pressures to tuberculosis. It is interesting that one patient, with marked mediastinal and cardiac retraction to the left, and with marked dyspnea on exertion, had normal venous pressure, although it is true that the pressure on the affected side was twelve points higher than that on the unaffected side. In speaking of the cardiac complications it might be well to discuss here the isolated hypertension of the lesser circulation found particularly in hemoptysis with prodromal symptoms preceding the hemorrhage. While we cannot deny the existence of such a syndrome, the overwhelming evidence in favor of the emotional influence on venous pressures, to be found in the study of our twenty-one cases during pneumothorax, would seem to offer adequate explanation of a rise in venous pressure in patients suffering from the prodromal symptoms of hemoptysis.

Pulmonary Complications.—In the patients with pleural complications of pulmonary tuberculosis (Table III), there were several interesting cases. It appears that sometimes, in tuberculous empyema, the venous pressure is elevated and, interestingly enough, not always on the affected side. Another surprising thing has been that patients, suffering from silicotuberculosis, appear to have a normal venous pressure. Certain French authors have found an increase of venous pressure in miliary tuberculosis, and one has tried to explain it on the basis of diffuse fibrosis. In silicotuberculosis we have every reason to expect a great deal more diffuse fibrosis than in miliary tuberculosis but, nevertheless, in the former cases, in our limited experience, and from what we can learn through personal communication with others studying this disease, the venous pressure is normal.

TABLE III

VENOUS PRESSURE IN PULMONARY COMPLICATIONS OF PULMONARY TUBERCULOSIS

COMPLICATION	VENOUS PRESSURE	
	RIGHT	LEFT
Tuberculous empyema, left	67	
Tuberculous empyema, left	126	138
Massive effusion, left	150	112
Silicotuberculosis	114	
Silicotuberculosis	90	

Bilateral Pneumothorax.—Whether the alleged increase of venous pressure following pneumothorax in certain forms of tuberculosis is due to a compression of the vascular bed of the lung or to local pressure on the right heart is a matter of dispute. The fact that in our patients with bilateral pneumothorax (Table IV) the venous pressures were nor-

TABLE IV

VENOUS PRESSURE AFTER THORACOPLASTY

SIDE OPERATED	EXTENT OF OPERATION	TIME ELAPSED SINCE OPERATION	VENOUS PRESSURE	
			RIGHT	LEFT
Left	Complete	20 months	68	150
Right	Complete	8 months	146	86
Right	Complete	4 years	90	
Right	Complete	1 year	100	66
Left	Complete with Schede	1 year	58	56
Right	6 ribs	6 weeks	66	66
Right	8 ribs	1½ years		140

mal with a high degree of pulmonary collapse would tend to be against the first explanation. Both of our patients had venous pressure determinations made on the left arm. In one, the degree of pulmonary collapse was 40 per cent on the right and 70 per cent on the left, and the venous pressure was 90. In the other there was a 50 per cent collapse on the right and 60 per cent on the left, and the venous pressure was 120. In three patients with initial pneumothorax the venous pressure

was normal and all received their insufflation on the right. In two of these the venous pressure readings were taken from the left arm and were 64 and 90, respectively. In the remaining patient the venous pressure reading was taken from the right and eventually stabilized at 50.

Unilateral Pneumothorax.—In the patients with unilateral pneumothorax (Table V), pressures above 120 mm. were found three times;

TABLE V

PNEUMOTHORAX CASES

SIDE OF PNEUMOTHORAX	SIDE OF VENOUS PRESSURE	MM. H_2O	PERCENTAGE OF COLLAPSE
Right	Right	61	65
Right	Right	104	85
Right	Right	88	80
Right	Right	54	70
Right	Right	120	75
Right	Right	50	50
Right	Right	86	75
Left	Left	70	70
Left	Left	138	100
Left	Left	99	85
Left	Left	120	60
Left	Left	94	65
Left	Left	70	80
Left	Left	42---Average 86	70
Right	Left	86	75
Right	Left	52	90
Right	Left	68	50
Right	Left	56	65
Right	Left	76	65
Right	Left	47	80
Right	Left	68	90
Right	Left	102	45
Right	Left	134	75
Right	Left	90	Initial pneumothorax
Right	Left	90	40
Left	Right	116	90
Left	Right	110	90
Left	Right	58	85
Left	Right	112	45—Fluid
Left	Right	50	65
Left	Right	96	80
Left	Right	195	75
Left	Right	67	75
Left	Right	92	90
Left	Right	120---Average 90	50

one of these, with a pressure of 138 on the pneumothorax side, had a quite rigid pleural space due to prolonged pleural exudate. We can offer no explanation for increased pressures of the others. On the whole, we have been astonished at the very normal venous pressures of this group; in fact, in two patients exhibiting the classical symptoms of intolerance to collapse, such as dyspnea, the pressures were perfectly normal, contrary to what other writers have found. Fourteen patients with venous pressure determinations and pneumothorax on the same side showed an average venous pressure of 86, while twenty-one patients in

whom the venous pressure determinations and pneumothorax were done on opposite sides, had an average venous pressure of 90. We have already shown that the average venous pressure of ambulant patients is 94 while that of patients on bed rest is 85. This is not a very marked difference and the evaluation of these figures is found complicated by the fact that the percentage of collapse in ambulant patients was 64 while that in patients on bed rest was 80.

TABLE V (A)
PNEUMOTHORAX CASES

SIDE OF PNEUMOTHORAX	SIDE OF VENOUS PRESSURE	MM. H_2O	PERCENTAGE OF COLLAPSE
Left	Left	138	100
Left	Right	110	90
Right	Left	52	90
Right	Left	68	90
Left	Right	116	90
Left	Right	92	90
Left	Left	99	85---Average 87%
Right	Right	104	85
Left	Right	58	85
Right	Right	88	80
Right	Left	47	80
Left	Right	96	80
Left	Left	70---Average 87	80
Right	Right	120	75
Right	Right	86	75
Right	Left	86	75
Right	Left	134	75
Left	Right	195	75
Left	Right	67	75
Right	Right	54	70
Left	Left	70	70---Average 71%
Left	Left	42	70
Right	Right	61	65
Right	Left	56	65
Right	Left	76	65
Left	Right	50	65
Left	Left	94	65
Left	Left	120---Average 87	60
Right	Right	50	50
Right	Left	68	50
Left	Right	120	50---Average 40%
Right	Left	102	45
Left	Right	112	45—Fluid
Right	Left	90	40
Right	Left	90---Average 90	Initial pneumothorax

The influence of the degree of pulmonary collapse on the venous pressure was found to be as follows: In seven patients having 50 per cent or less collapse (averaging 40 per cent), the venous pressure was found to average 90; in fifteen patients whose percent of collapse ranged from 60 to 75 (averaging 71 per cent), the venous pressure averaged 87; in thirteen patients having 80 to 100 per cent collapse (averaging 87 per cent), the venous pressure was 87.

Thoracoplasty Patients.—Seven patients who had had thoracoplasty (Table IV) were available for study. It is interesting that two of these

still showed an elevation of venous pressure on the side of the operation, one twenty months and one eight months after operation. In another, although the venous pressure was normal, that of the side of the operation was 34 mm. higher than the contralateral side, one year after thoracoplasty. Interestingly enough the patient who had had the most extensive thoracoplasty, with a Schede, one year previously, had rather low venous pressure on both sides. In one patient in whom, due to technical difficulties, we were unable to get the venous pressure on the side of the operation, the pressure on the contralateral side was 140 mm. These facts were interesting in the light of the work reported by Overholt and Pilcher. If these high pressures were taken as a contraindication to operation, these patients who were in perfectly good clinical condition would be refused operation.

Simultaneous Arterial, Venous, and Pleural Pressures and Volume of Air.—It was possible, in the manner described above, to determine simultaneously the venous, arterial, and pleural pressures and volume of air given in twenty-one cases; so far as we have been able to find in the literature this is the first time that such a study has been made. A study of these cases is interesting in that it shows a marked effect of emotional tension and pain on venous pressure prior to the insertion of the pneumothorax needle, but an astonishing lack of influence of the actual insufflation of air upon the venous pressure, regardless of the volume of air injected and regardless of the initial and final pleural readings.

A division of these patients into those who were on strict bed rest as opposed to the group which were ambulant, or partially ambulant, showed the pressure variation in the two groups to be entirely alike. In one patient from whom air was removed, even the sudden decompression of the pleural cavity from +10, +15 to –2, +8 was accompanied by a drop of only 14 mm. of water in a curve which was falling from 139 at the beginning to 52 at the end. Whether or not this fall of 14 points during decompression is significant in such a curve seems very doubtful.

Analysis on Full Inspiration.—On full inspiration the venous pressure in eleven patients fell from 2 to 20 mm., an average of 5 mm. In four patients the venous pressure rose from 8 to 14 mm., an average of 12 mm. The average fall in venous pressure on full inspiration for all patients was 0.5 mm. The systolic pressure in eight patients fell from 2 to 10 mm., an average of 7.5. In three patients it rose from 2 to 18 mm., an average of 10. The average increase in systolic pressure for the entire group during full inspiration was 3 mm. The diastolic pressure in five patients fell from 4 to 10 mm., an average of 8. In seven patients it rose from 2 to 10 mm., an average of 5. The average decrease in diastolic pressure for the entire group was 0.16 mm.

Full Expiration.—On full expiration the venous pressure in eight patients rose from 4 to 40 mm., an average of 10. The remainder of

the patients showed no essential change. The systolic pressure in three patients dropped from 2 to 8 mm., an average of 5. In six patients it rose from 4 to 12 mm., an average of 8. The average increase for the entire group was 2.6 mm. The diastolic pressure fell in only two patients, 2 and 4 mm. respectively. In eight patients it rose from 2 to 20 mm., an average of 8. The average increase in diastolic pressure for the entire group was 5 mm.

Anesthesia Needle in the Skin.—While the anesthesia needle was piercing the skin and infiltrating the subjacent tissue, the venous pressure in three patients fell from 4 to 10 mm., an average of 7. In eight patients it rose from 2 to 20 mm., an average of 8. The increase in venous pressure averaged 2 mm. The systolic pressure in six patients fell from 2 to 10 mm., an average of 5. In seven patients it rose from 2 to 10 mm., an average of 5. For the entire group there was no essential change in the average systolic pressure. In nine patients the diastolic pressure fell from 2 to 18 mm., an average of 6. In three patients it rose from 2 to 6 mm., an average of 4. The average decrease in diastolic pressure was 2.5 mm.

Anesthesia Needle Through the Pleura.—When the anesthesia needle pierced the pleura the venous pressure in six patients fell from 2 to 10 mm., an average of 5 mm. In three patients it rose from 2 to 6 mm., an average of 3. The average fall for the entire group was 1 mm. The systolic pressure in fifteen patients fell from 2 to 20 mm., an average of 5 mm. The diastolic pressure in eleven patients fell from 2 to 12 mm., an average of 7 mm. In three patients it rose from 2 to 4 mm., an average of 3. The average fall in diastolic pressure for the entire group was 3.3 mm.

Pneumothorax Needle Through the Pleura.—In putting the pneumothorax needle through the pleura, particularly the larger needles, there is considerable emotional and muscular tension on the part of the patient so that it is difficult to discount these factors, the latter of which may reproduce the Valsalva experiment of breathing against a closed glottis. The venous pressure in five patients fell from 2 to 12 mm., an average of 8. In three patients it rose from 4 to 60, an average of 24. There is little doubt that the patient whose venous pressure rose 60 mm. produced this by an apprehensive catch of the breath and closing of the glottis which essentially reproduced the Valsalva experiment. The average increase of venous pressure for the entire group was 1.6 mm. The systolic pressure in seventeen patients fell from 2 to 12 mm., an average of 7. In one patient it rose 4 mm. The average fall in systolic pressure for the entire group was 5.5 mm. The diastolic pressure in ten patients fell from 2 to 10 mm., an average of 6.2. In four patients it rose from 4 to 10 mm., an average of 8. The average fall in diastolic pressure was 2 mm. These results on diastolic and systolic pressure, as well as all other results which followed needling the pleural cavity, are

entirely comparable with those previously published by Steidl and Heise,[14] although the changes are not quite so marked in degree.

Pneumothorax Needle Out.—The venous pressure in four patients dropped from 2 to 4 mm., an average of 3 mm., when the needle was withdrawn. In one patient it rose 2 mm. The average change for the group was practically zero. The systolic pressure in ten patients fell from 4 to 10 mm., an average of 5.8. In two patients it rose 2 and 4 mm., respectively. The average decrease for the entire group was 2.5 mm. The diastolic pressure in seven patients fell from 2 to 45 mm., an average of 10.7. In four patients it rose 2 mm. each. The average fall for the entire group was 3 mm.

Volume of Air Given.—Not considering two patients from whom small volumes of air were withdrawn, the volume of air given varied from 100 to 600 c.c., an average of 379 c.c.

Average Pleural Pressure Before Pneumothorax.—The mean pleural pressure before pneumothorax varied from –3 to +10. The average mean pleural pressure before pneumothorax was ÷1. The mean pleural pressure after pneumothorax varied from –9 to +12. The average mean pleural pressure after pneumothorax was +3.7.

Average Venous Pressure at Beginning of Experiment.—The venous pressure at the beginning of the experiment varied from 40 to 220 mm.; seven of these were above 120. It is important to remember that these patients were all under basal conditions before the experiment started and obviously the mere exhibition of needles disturbed these conditions. The average venous pressure for the entire group before the experiment was 113.

Average Venous Pressure Just Before Pneumothorax Treatment.—The venous pressure just before the injection of air varied from 50 to 120 mm., an average of 85. This drop is interpreted as a mental and physical relaxation of the patient after needling.

Average Venous Pressure After Insufflation.—The venous pressure after the pneumothorax was completed varied from 50 to 120 mm., an average of 83. It is thus apparent that the actual process of insufflation of air into the pleural cavity had no appreciable effect upon the venous pressure in our series of cases; this we regard as the important point of this study.

Venous Pressure Taken Simultaneously at the Time of Pneumothorax.—In addition to the above group, a small series of five patients had their venous pressures taken continuously during the administration of pneumothorax and without a simultaneous study of the arterial pressures. The venous pressures of these patients did not differ materially from those discussed in the previous group. Of the five, three showed definitely elevated venous pressure at the beginning of pneumothorax, respectively, 144, 150, 204. The patient with a pressure of 150 was constantly at rest in bed; only one of these five patients showed an eleva-

tion above normal at the end of pneumothorax and this was the patient whose pressure at the start was 150. All five showed lower pressures after pneumothorax than when first taken. This change varied from 12 to 92 mm. of water. It is apparent, therefore, that the venous pressures of these patients reacted in a similar manner to that of the larger group.

DISCUSSION

In considering this work as a whole, several facts stand out. One is the instability of the venous pressure under the influence of emotional factors. Except for descriptions of basal conditions this fact, so far as we know, has not been commented upon in the literature; in fact, Fuchs[16] commented upon his finding the venous pressure, even in nervous and hysterical women, ranging from 100 to 120 mm. and, in spite of reassurance from the doctor, the pressure did not fall. For years attention has been focused on the influence of emotion upon arterial pressure and it has been astonishing to find that its effect is relatively far greater on venous pressure. It has also been surprising to find the venous pressure so normal in patients in whom other authors have found it abnormal, such as the cardiac complications, generalized diffuse fibrosis, and symptoms of intolerance to compression. That nervous and emotional factors, rather than physical activity, disturb these basal conditions is amply apparent by the division of the group into those who are and have been on complete rest, seventeen, and those who are more or less ambulatory, twenty. In the former group the average venous pressure was found to be 85 (with two definitely elevated and one a borderline case); the degree of pulmonary collapse in this group was 80 per cent. In the ambulatory group the average venous pressure was 94 (one definitely elevated and three borderline cases), and the average degree of collapse was 64 per cent.

The prolonged period during which there is a unilateral elevation of pressure in thoracoplasty cases has also been surprising. The few instances in which the venous pressure was found elevated have demonstrated the necessity of studying the pressure in both arms in tuberculosis. It is difficult to understand why previous students of this subject have not emphasized this point because all are agreed that when elevation of venous pressure occurs in pulmonary tuberculosis, it is the result of local mechanical factors (which must vary from side to side) as opposed to elevations due to cardiac insufficiency in which elevation of venous pressure is generalized.

CONCLUSIONS

Venous pressure is rather rarely elevated in pulmonary tuberculosis even in collapse therapy and much less influenced by mechanical than by emotional factors. Fluid, pneumothorax, and thoracoplasty may cause elevation of venous pressure but rather rarely, and usually such

elevation is unilateral. The actual insufflation of air into the pleural cavity does not disturb the venous pressure. The elevations following thoracoplasty may be present for long periods of time and, therefore, are not of absolute value as a contraindication to operation. It would seem that determination of venous pressure is of value in a few patients with pulmonary tuberculosis, but not of value as a routine sanatorium procedure with all patients.

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STUDIES OF THE JUGULAR, CAROTID, AND PULMONARY PRESSURES OF ANESTHETIZED DOGS DURING POSITIVE INFLATION OF THE LUNGS*

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MAINTENANCE of respiration by positive inflation of the lungs has made possible the rapid advance in technique of thoracic operations which has occurred in recent years. The effects of this expedient on the circulation, however, have not received much attention. It has been shown experimentally that the amount of blood pumped each minute through the heart of anesthetized dogs falls as the pressure of gas within their lungs is increased,[1] but the mechanism which results in this circulatory impairment is not completely understood. If the risk of intrathoracic operations is to be minimized, it would seem that a fuller understanding of this mechanism is important.

It is possible to attribute the reduction of cardiac output which accompanies insufflation of the lungs to two factors: (1) compression of the heart and its afferent vessels which interferes with filling and (2) compression of the pulmonary vessels which interferes with emptying. Although the latter is frequently considered to be the chief danger of insufflation the former seems to us the more likely to influence cardiac output because of the known ability of the right heart to overcome resistance to its emptying. In previous experiments we were unable to distinguish between the effects of these two factors by direct measurements of cardiac output under various conditions. The experiments here presented were designed therefore to attack the problem indirectly by means of blood pressure studies.

METHOD

Dogs anesthetized by the intravenous injection of barbital sodium were used. Some of the observations were made on intact animals; others were made after the chest of the animal had been opened wide by splitting the sternum and introducing a self-retaining retractor. The lungs were insufflated with oxygen by means of a metal intratracheal cannula, through which a rubber catheter was passed loosely into the trachea. The degree of inflation was controlled by means of a screw clamp applied to the expiratory limb of the cannula. The venous pressure was measured by the apparatus of Moritz and von Tabora[2] connected to a cannula in the external jugular vein. The leveling arm of the apparatus was adjusted to the level of the bony prominence of the shoulder of the dog as it lay on its back. This seemed to us a more accurate method of approximating the level of the base of the heart

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than using the sternum, as is done with human measurements. It is, however, obviously liable to considerable error, so that the readings cannot be considered accurate in absolute value; the relative change, and therefore the shape of the curve, is not influenced by this error, and is the finding which concerns us.

In those experiments, in which systemic and pulmonary artery pressures were studied, direct kymographic tracings were obtained by means of simplified tambour manometers of the Hürthle type, connected to cannulas in the common carotid artery, and in one of the secondary branches of the left pulmonary artery respectively.

EXPERIMENTS

In four experiments (No. 135, 136, 137, and 138) a series of venous pressure readings were obtained while the lungs of a dog, whose chest had been opened wide, were inflated at varying pressures. These experiments established the fact, confirmed by all later ones, that a rise in intratracheal pressure is always associated with a rise in jugular venous pressure. The venous pressure did not rise in direct proportion, but when plotted graphically against the intratracheal pressure followed a characteristic curve. Wide individual variations in steepness and height, however, were found to occur in different animals, so that each curve has been charted separately (Fig. 1).

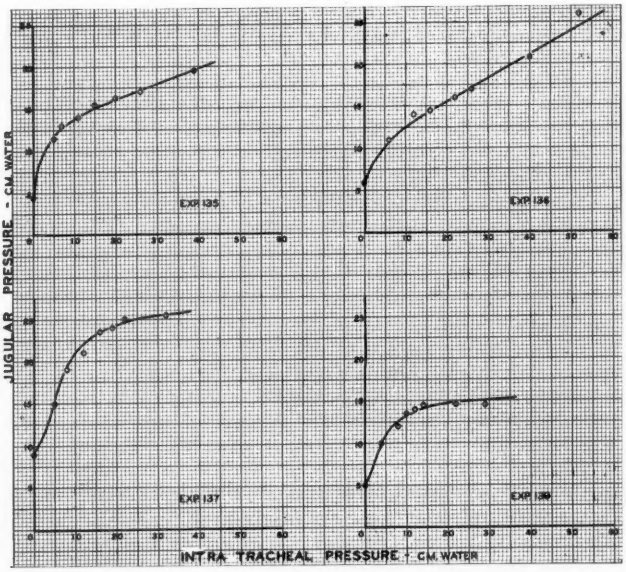

Fig. 1.—Venous pressure readings in the first four experiments while the dog, with chest wide open, was kept alive by continuous intratracheal insufflation at varying pressures, plotted graphically against the intratracheal pressure.

It was thought that if this rise in venous pressure were due to compression of the heart as, we suspected, the curve would be steeper in a closed chest than in an open one; if, on the other hand, the rise in venous pressure were due simply to resistance in the pulmonary circuit, caused by pressure within the alveoli, this would not be the case. Therefore, the studies were repeated in *intact* animals.

In this, the second group of our experiments, respirations were paralyzed by the administration of curare. This was found to be necessary because when the intra-

tracheal pressure of animals breathing normally was raised, their respiratory efforts caused such wide and irregular fluctuations in intratracheal pressure that the relationship between intratracheal pressure and venous pressure was almost impossible to determine. Observations were made on six animals (No. 140, 141, 142, 143, 144, and 145), first with chest closed and then with chest open. In every experiment the venous pressure curve was found to rise more rapidly when the chest was closed, which resulted in a crossing of the curves in some of the experiments (Fig. 2). These results supported our belief that the rise in venous pressure which accompanies insufflation of the lungs is due to compression of the heart and not to resistance in the pulmonary circuit.

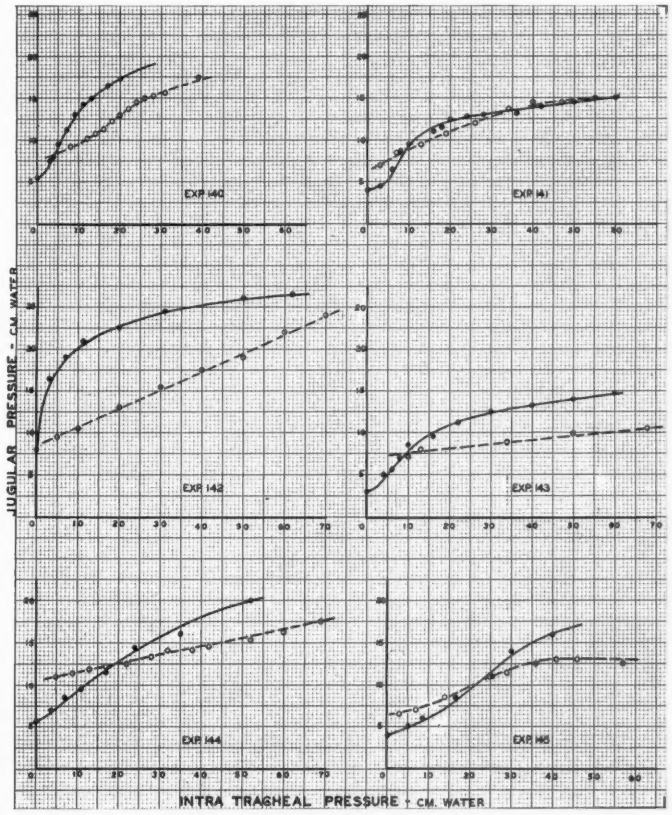

Fig. 2.—Venous pressure readings in the second group of experiments, plotted graphically against intratracheal pressure. After curarization, observations were made first with the chest closed (dots) and then with the chest open (circles). The venous pressure curve is shown to rise more rapidly when the chest is closed, resulting in a crossing of the curves in some experiments.

In order to put this conception to a further test, it was thought desirable to study the blood pressure in the pulmonary and systemic arteries (as well as the venous pressure) during increasing intratracheal pressure. Accordingly, four animals (No. 150, 151, 152, and 153) were curarized and in each instance, after venous pressure readings had been obtained at varying intratracheal pressures, the chest was opened,

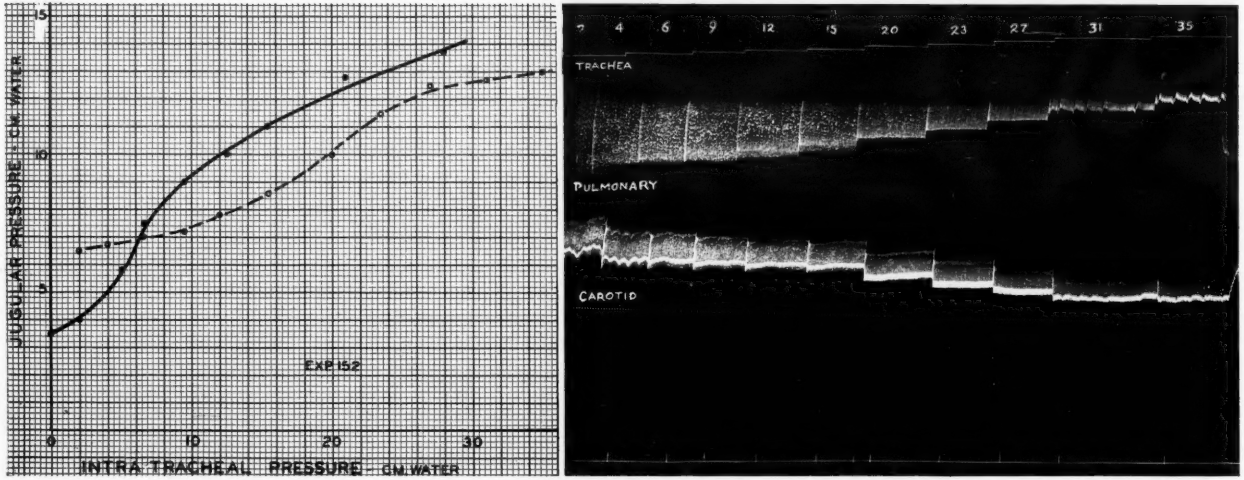

Fig. 3A.

Fig. 3.—*A*, *B*, *C*, and *D*, venous pressure readings in the third group of experiments, plotted graphically against intratracheal pressure, with the kymographic records of intratracheal, carotid and pulmonary artery pressures made after the chest had been opened. When intratracheal pressure was raised, pressure in the carotid artery fell, while that in the pulmonary artery rose.

a cannula was introduced into one of the secondary branches of the left pulmonary artery, and a second series of venous pressure readings at increasing intratracheal pressures was obtained. At the same time kymographic records of intratracheal, carotid, and pulmonary artery pressures were made. These tracings showed that when intratracheal pressure was raised, pressure in the carotid artery fell, while that

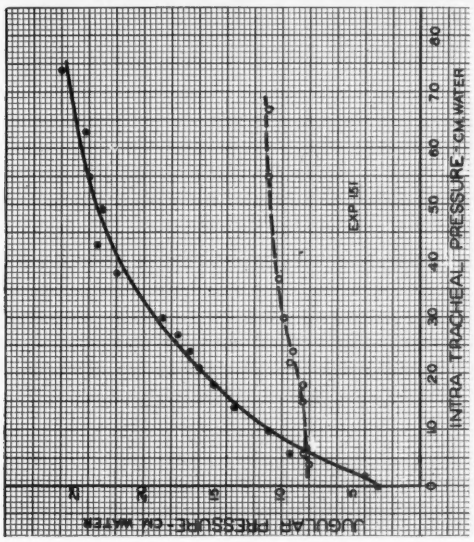

Fig. 3*B*.

in the pulmonary artery rose, and that pulmonary diastolic pressure rose more rapidly than systolic, with resultant fall in pulse pressure in most cases (Fig. 3). A real obstruction in the pulmonary circuit was thus demonstrated to have been present.

In order to compare these blood pressure changes with those of known pulmonary artery obstruction, two more experiments were done (No. 155 and 156). In these

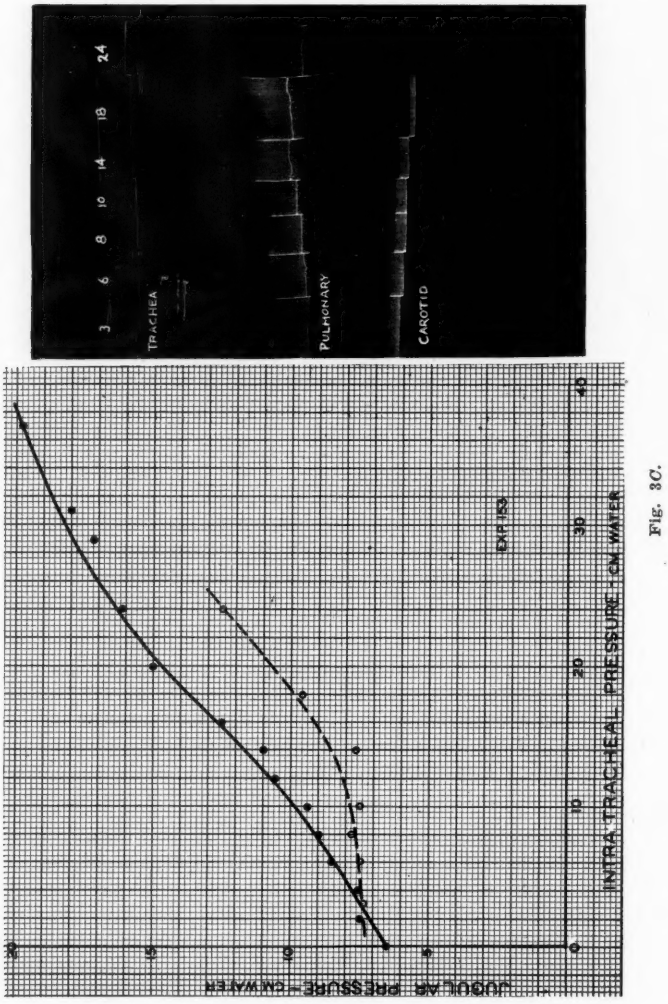

Fig. 3C.

the chests of curarized animals were opened wide and intratracheal, carotid, and pulmonary artery pressures were recorded on a kymograph. Tracings were obtained first during a period of increasing intratracheal pressure, and then during a period in which the right pulmonary artery was gradually occluded by means of a lobectomy tourniquet around the root of the lung. These tracings showed that when the

tourniquet was tightened, the pulmonary systolic pressure rose as high as it did during high intratracheal pressure, but that, unlike the effect of high intratracheal pressure, the pulse pressure increased, and the systemic arterial pressure was not appreciably affected (Fig. 4).

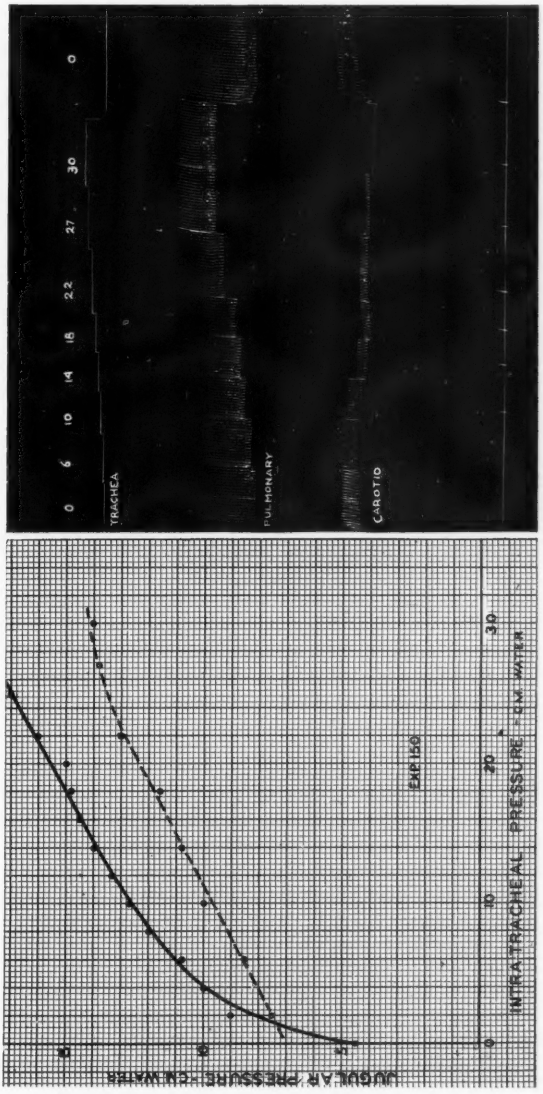

Fig. 3D.

How these findings confirm our conception of the primary importance of interference with the filling of the heart is discussed further.

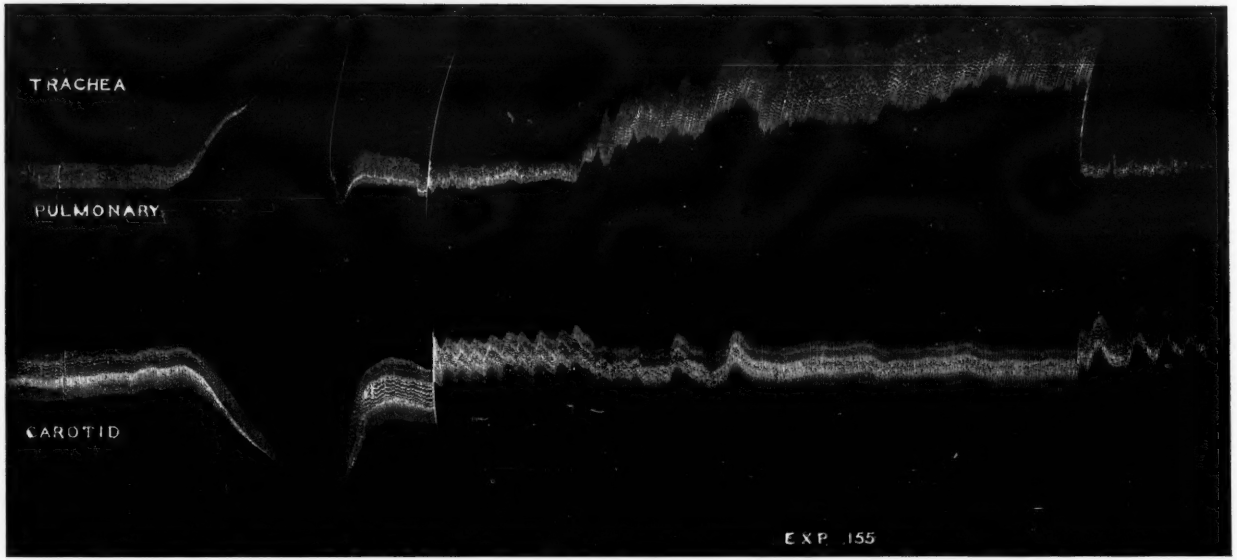

Fig. 4A.

Fig. 4.—*A* and *B*, kymographic records of intratracheal, carotid, and pulmonary artery pressures showing, first, a period of increasing intratracheal pressure, and then a period in which the right pulmonary artery was gradually occluded by means of a lobectomy tourniquet around the root of the lung. When the tourniquet was tightened, the pulmonary systolic pressure rose as high as it did during high intratracheal pressure, but the pulse pressure increased, and the systemic arterial pressure was not appreciably affected.

DISCUSSION

Half a century ago Cohnheim[3] showed that an increased pressure in the pericardial cavity of a curarized dog caused by the injection of oil is accompanied by a rise in pressure in the jugular vein and a fall in

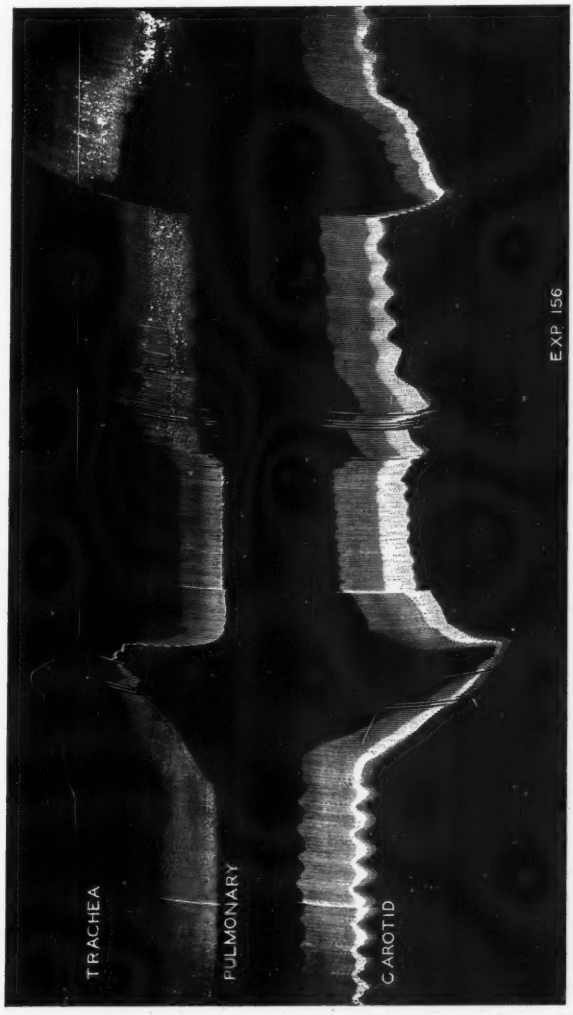

Fig. 4B.

pressure in the femoral and pulmonary arteries. This is the phenomenon which had already been described by Rose[4] and named by him *Herztamponade*. The rise in venous pressure and fall in arterial, characteristic of tamponade, was a constant finding in our experiments. *Was this*

finding due to a tamponade effect caused by compression of the heart (which hinders its filling), or was it the effect of increased resistance in the pulmonary circuit (which interferes with the emptying of the heart)? In our experiments the curve of venous pressure showed a more abrupt rise when the chest was closed than when the chest was open. Since at any given intratracheal pressure the resistance in the pulmonary circuit is no greater when the chest is closed than when the chest is open, the more abrupt rise in the venous pressure curve which we have observed in the closed chest suggests that cardiac compression was present. By assuming its presence one can explain the fact that at very low intratracheal pressures the venous pressure was often higher when the chest was open because the elastic recoil of the lungs had not been completely overcome and the heart was still exposed to some degree of negative pressure in the closed chest whereas in the open chest the heart was exposed to atmospheric pressure. At moderate intratracheal pressures the venous pressure was always lower when the chest was open because the pressure of the distended lungs on the heart was less effective than when the chest was closed. At high intratracheal pressures the venous pressures sometimes differed less than at moderate ones because the lungs bulged from the open chest and exerted almost as much pressure on the heart as when the chest was closed.

It seems probable, then, that a definite tamponade was produced when intratracheal pressure was raised. But, in the experiment described by Cohnheim, the pressure in the pulmonary artery fell, whereas in these experiments it rose. Thus when intratracheal pressure was raised, the resistance in the pulmonary circuit must also have been increased. Was this increase in resistance sufficiently great to have an influence on cardiac output?

In this connection Cohnheim[3] wrote: "The physiological heart muscle can meet the demands on its work or it cannot meet them: in the former case we have regular physiological circulation, in the latter, death." He came to this conclusion by gradually constricting a loop passed around the pulmonary artery of a dog and observing that no change occurred in femoral or jugular pressure until a critical point was reached. Since then it has been shown that up to 60 per cent of the pulmonary outlet can be blocked without affecting systemic arterial or venous pressures,[5, 6] or cardiac output.[7] Yet the cardiac output often falls when the intratracheal pressure is raised relatively little,[1] and the experiments here reported show slight increases in intratracheal pressure to be accompanied by the peripheral phenomena of tamponade. Furthermore, the last two experiments demonstrate, as Cohnheim and, later, Underhill did, that the pulmonary arterial pressure may be raised considerably by occluding all the branches to one lung, without any accompanying fall in systemic pressure, or any drop in output.[8] A comparison of the tracings reveals, further, how this may take place. For

when the pulmonary arterial pressure is raised by tieing off one lung, the blood returning unobstructed to the heart is forced vigorously by the heart through the vessels which remain open, producing a high pulse pressure; but when the pulmonary arterial pressure is raised through distention of the alveoli the systolic contraction of the right heart cannot raise the pulmonary systolic pressure much above the diastolic, because the supply of blood returning to it is inadequate. This failure of the heart to fill must then be the chief factor in causing the cardiac output to fall when the lungs of animals or patients are insufflated at too high a pressure.

It seems evident that the height to which the intratracheal pressure can be raised, without causing appreciable circulatory impairment, varies in different individuals, and in the same individual at different times, in accordance with their ability to compensate for the increased intrathoracic pressure by a corresponding increase in venous pressure (in order to maintain the effective venous pressure). The compensatory rise in venous pressure depends on several factors, the more important of which perhaps are an adequate circulating blood volume, and a veno-pressor effect, the importance of which has been stressed by Henderson[9] but the nature of which is not understood. When either of these factors is reduced, the ability of a patient to compensate for a tamponade effect is likewise reduced, and an intratracheal pressure which under normal conditions would be safe might prove dangerous.

It seems evident also, that, while it is compression of the heart which is ordinarily the main factor in impairing circulation during insufflation of the lungs, a definite added burden is put on the right ventricular muscle because of an increased resistance in the pulmonary circuit. If this resistance is still further increased by the operative procedure, such as ligation of a large branch of the pulmonary artery, the heart may no longer be able to overcome it especially if any myocardial weakness be present.

SUMMARY

Sixteen experiments on anesthetized dogs are reported. In a group of four, venous pressure readings were obtained while the lungs of the animals with chests wide open were insufflated at varying pressures. In a group of six, following curarization, venous pressure readings during insufflation at varying pressures were obtained before and after opening the chest. In another group of four curarized animals (in addition to venous pressure readings) tracings of pulmonary and carotid artery pressures were obtained after opening the chest. In two other open chest experiments the changes in pulmonary and carotid artery pressures which accompany insufflation at increasing pressures were contrasted with the changes in pulmonary and carotid artery pressures which follow ligation of the root of one lung.

The findings were as follows:

1. A rise in intratracheal pressure was always associated with a rise in venous pressure.

2. As the intratracheal pressure was increased venous pressure rose more rapidly when the chest was closed than when it was open.

3. When intratracheal pressure was raised, pressure in the carotid artery fell, while that in the pulmonary artery rose.

4. The rise in pulmonary artery pressure which accompanied insufflation of the lungs at increasing pressure was usually associated with a decrease in pulmonary pulse pressure, whereas the rise in pulmonary artery pressure which followed ligation of the right lung was always associated with an increase in pulmonary pulse pressure.

We believe these findings indicate that the decrease in cardiac output which has been shown to accompany positive inflation of the lungs is due to compression of the heart which hinders its filling rather than to compression of the pulmonary vessels which interferes with its emptying.

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A PULMONARY ARTERY ARISING FROM THE ABDOMINAL AORTA

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ACCESSORY pulmonary arteries have been regarded formerly as anatomic curiosities, and their main interest centered about the rarity of their occurrence and their possible embryologic significance. However, with the development of modern thoracic surgery, and particularly the operations of lobectomy and pneumonectomy, such anomalies take on a new significance.

McCotter, in 1910, reported the occurrence of an artery, 7 mm. in diameter, which arose from the thoracic aorta, on a level with the tenth thoracic vertebra, 7 cm. above the coeliac axis, and which passed upward and to the right between the folds of the pulmonary ligament to the inferior medial margin of the right lung. A review of the literature at that time revealed only nine recorded cases of accessory pulmonary arteries from the thoracic aorta, one from the abdominal aorta, and one from an intercostal artery. Since then three additional cases have been reported by Park, Doering, and Ingalls. In his report Park calls attention to Eppinger's case of a pulmonary artery arising from the right phrenic artery.

The anomaly herewith described is the second to be reported. In 1802 Maugars described a pulmonary artery arising from the abdominal aorta just superior to the coeliac axis. This artery passed upward through the esophageal hiatus of the diaphragm and then divided into two branches supplying the inferior lobe of each lung. This occurred in a 7-year-old child who showed no other abnormalities upon autopsy except increased dimensions of both lungs and both kidneys. Death was attributed to a nervous affection or fever.

The present case was observed during the routine dissection of cadavers in the student laboratory. The subject was a well-developed, white male, aged 40 years. The cause of death was given as general paralysis and heat stroke. Upon removal of the right lung from the thorax, a large artery, 8 mm. in diameter, was seen entering the medial, inferior border of the right lung. This artery measured 9 cm. in length and was not accompanied by any other structures. It appeared to pierce the diaphragm through a hiatus of its own. This hiatus was on a level with the esophageal opening and about 1 cm. medial to it. Subsequent dissection of the abdomen revealed the origin of this anomalous vessel to be the upper part of the coeliac axis. In addition to this

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pulmonary branch, the coeliac axis gave off a right and left phrenic, and the usual left gastric, hepatic, and lienal arteries. The accessory pulmonary artery was the largest branch, whereas the others were quite normal in size and distribution. The coeliac axis was twice the normal size. There were no other anomalies of the thoracic or abdominal aorta. The heart was normal. However, there was an aneurysm of the ascending portion and arch of the thoracic aorta measuring 6 cm. in diameter. Examination of the lungs revealed findings typical of a bilateral confluent, lobular pneumonia and a right-sided empyema.

Fig. 1 shows the origin, course, and relations of the accessory pulmonary artery. The diaphragm has been divided anterior to the artery, showing the esophageal hiatus to the left at the same level, the hiatus for the inferior vena cava superiorly and to the right, and the aortic hiatus posteriorly and below. All the mediastinal structures have been removed with the exception of the accessory pulmonary artery entering the inferior lobe of the right lung.

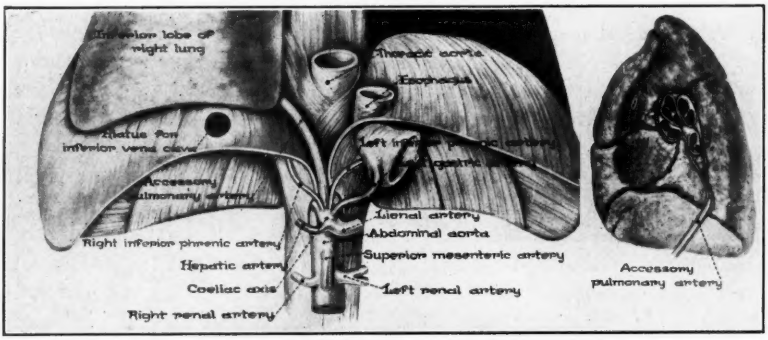

Fig. 1.—This illustrates the origin, course, and relations of the accessory pulmonary artery. The diaphragm has been divided at its sternal attachment straight posteriorly to the hiatus for the accessory artery. The esophageal hiatus is to the left at the same level, the hiatus for the inferior vena cava superiorly and to the right, and the aortic hiatus posteriorly and below. The coeliac axis gives rise to six arteries: the accessory pulmonary, the right and left inferior phrenics, the left gastric, hepatic, and lienal arteries. Normally there are only three divisions, the inferior phrenics coming off independently from the abdominal aorta above the coeliac axis. The median sagittal view of the right lung shows the anomalous vessel entering between the folds of the pulmonary ligament.

Fig. 2 is an x-ray photograph of the right lung showing the area supplied by the anomalous artery by means of the injection of a solution of red lead. It is evident that the accessory artery supplied only the posteroinferior portion of the inferior lobe. The smaller vessels radiating from the hilum of the lung are the bronchial arteries which were injected with the general arterial system when the cadaver was prepared for dissection. Such an injection, of course, does not reach the right heart or the pulmonary arteries.

Besides its obvious surgical significance this case suggests several interesting considerations. Embryologically it is an event difficult to

explain. Fedorow believed that the pulmonary artery grew as a bud directly from the sixth aortic arch to inosculate, after dividing into many branches, with the plexus around the pulmonary anlage, while the vein developed in a similar manner from the sinus venosus to inosculate with the opposite extremity of the plexus. Such a view does not allow an explanation of the anomaly which has been described. Thoma, Evans, and Brown have presented the commonly accepted

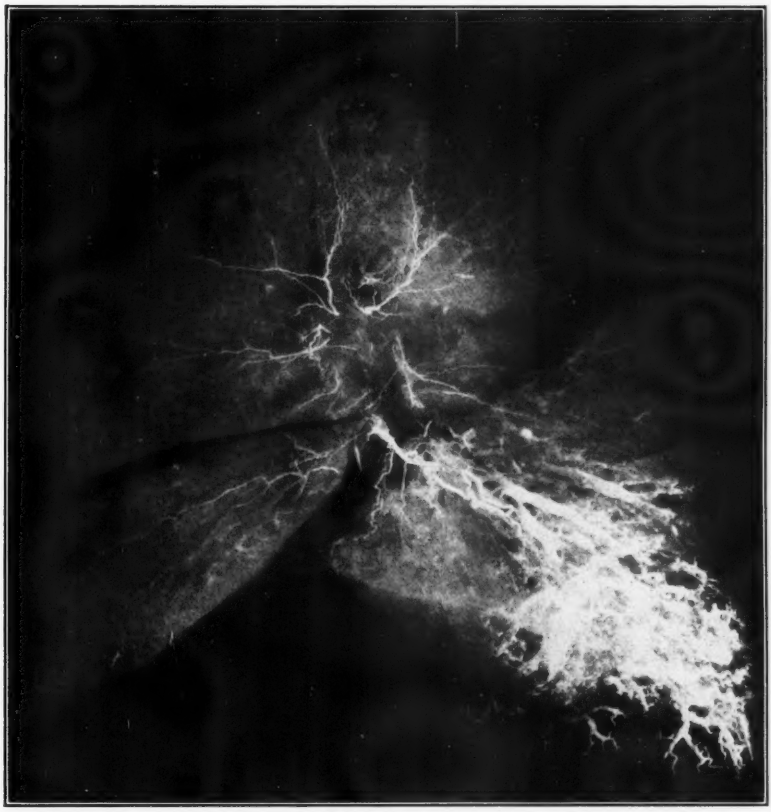

Fig. 2.—An x-ray photograph of the right lung showing the area supplied by the anomalous artery by means of the injection of a solution of red lead. The accessory artery supplies only the posteroinferior surface of the inferior lobe. The smaller vessels radiating from the hilum of the lung are the bronchial arteries which were injected with the general arterial system when the cadaver was prepared for dissection; such an injection, of course, does not reach the right heart or the pulmonary arteries.

teaching which states that the blood vascular system develops as a capillary plexus (area vasculosa) which spreads in all directions. Out of this plexus channels develop which subsequently become arteries or veins, depending upon whether they are developed on the arterial or venous side. The plexus around the lung bud, according to Brown, is

differentiated into two systems: (1) from its ventral and caudal portion, the pulmonary for the fulfillment of the respiratory function, and (2) from the caudal and cephalic portion, the bronchial for the nutrition of the lung tissue proper. That portion of the splanchnic plexus between the pulmonary anlage cephalad and hepatic sinusoids caudad serves as a direct temporary communication between the pulmonary and systemic circulations. With the further development of the pulmonary vessels these communications disappear. The persistence of one of these channels is sufficient explanation for the development of an accessory pulmonary vessel. The far more common occurrence of small arteries extending laterally from the thoracic aorta between the folds of the pulmonary ligament to the lung, as observed by Turner, can be explained on the same basis. The fact that the accessory pulmonary artery pierces the diaphragm presents no special difficulties, as the transverse septum develops much later, and invests the anomalous structure in much the same way as the aorta, the inferior vena cava, and the esophagus.

Blood in the systemic aorta contains oxygenated hemoglobin. After such blood passes through the lung capillary beds surrounding the alveoli, it leaves the lung in much the same condition in which it enters the lung. It follows, then, that the area of lung supplied by the accessory pulmonary artery is nonfunctioning tissue. This is a possible explanation for the enlargement of the lungs in Maugar's case, the increase in size being compensatory for the tissue in both lungs lost to respiration.

The blood pressure within the pulmonary circulation is approximately one-sixth that within the systemic circulation (Starling). A direct communication between the two circulations results then in a true, life-long, pulmonary hypertension. If, as Moschowitz states, a sustained hypertension of the lesser circulation leads to arteriosclerosis of the pulmonary vessels, there should be a marked difference between the walls of the anomalous pulmonary artery described and the pulmonary arteries entering the lung at the hilum. Accordingly, microscopic sections were made of tissue supplied by the anomalous vessel and of tissue from the upper portion of the same lobe. The report by Dr. C. V. Weller was as follows: "This tissue is very poorly fixed so that nuclear staining is impossible. There is intense pulmonary congestion with some hemorrhage. The anomalous pulmonary artery shows some sclerosis of its wall with medial calcification which is not present in the other section."

This observation is extremely interesting as it constitutes an absolutely unique verification of the widely held view that prolonged arterial hypertension is followed by sclerotic changes in the vessel walls.

The author wishes to express his sincere appreciation to Dr. Rollo E. McCotter, professor of anatomy, who granted permission to report this case from his laboratory.

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ACTINOMYCOSIS THORACIS

Report of Two Arrested Cases

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AVAILABLE statistics place the incidence of thoracic involvement in actinomycosis from 7 to 23 per cent and indicate that in approximately half of these instances the disease apparently is confined to the thorax. Because there have been reported so few "arrested" or cured cases in this group and because there prevails a rather hopeless attitude in regard to the prognosis of systemic actinomycosis in general, two cases which have remained well, sixteen and twenty-six months following final closure of their operative wounds, are reported. In both cases the infection involved the pleural cavity and chest wall and in one the empyema cavity communicated with a bronchus through a small superficial pulmonary abscess.

The treatment of both cases consisted primarily of an attempt to eradicate the disease by as complete an excision of all infected tissue as possible in addition to the conventional therapy of massive doses of iodine and roentgen irradiation. Preliminary drainage of the chest wall and empyema cavity was established, and as soon as possible (from two to four weeks later) was followed by complete saucerization of the empyema cavity by Schede thoracoplasty.

Ten days later in one case and twenty-eight days in the other one, the infected tissue was excised as widely as possible by means of surgical diathermy, removing a layer from the entire surface lining the cavity, including pleura, lung tissue, and chest wall. After granulation tissue had reappeared over the surface this procedure of electro-surgical debridement was repeated once in one case and twice in the other one. In the latter case the third procedure was carried out because actinomyces were cultured from granulations which developed after the first excision. In both cases no fungi were found in numerous subsequent smears and cultures from the surface and from granulation tissue removed from scattered areas. This however has little significance since actinomyces have no respect for tissue or inflammatory barriers and notoriously are absent from the surface and superficial tissues while present in deeper tissues and remote burrows where invasion is in progress. The frequency with which actinomycosis recurs locally or burrows and appears in regions remote to the primary site weeks, months, or even years after healing has taken place and an apparent cure effected, makes the pro-

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Read (by title) before the Twentieth Annual Meeting of the American Association for Thoracic Surgery, May 31, June 1 and 2, 1937, at Saranac Lake, New York.

nouncement "cured" a hazardous statement. For this reason both cases reported below have been classified, "arrested," although there is no evidence of residual disease to date.

CASE HISTORIES

CASE 1.—F. J., a carpenter, white male, aged 39 years, was seen in consultation and referred to the hospital Dec. 12, 1934. He complained of pain at the base of the right side of his chest, and a red tender mass just below the angle of the scapula. He had been well until five weeks previously when he developed a diarrhea which lasted about thirty-six hours. Following this he felt weak and tired and believed that he had some fever but continued to work. A week later he developed a pleurisy on the right side. His doctor strapped his chest and told him that he had some fever. He continued to have pain, though less severe, in his right chest, to complain of weakness and anorexia, and to lose weight. For one week he had had a dry cough and for four days had observed the development of a tender mass below his right shoulder blade. There was no history of association with infected animals. The family and past histories were unimportant.

The patient was tall, slender, and well developed. There was evidence of recent weight loss and a definite pallor of the skin and mucous membrane. His eyes appeared to be slightly sunken. On admission the pulse was 108, temperature 101.5, and respirations 28. There were several carious teeth and considerable pyorrhea.

The right side of the chest moved very little with respiration and in the mid-scapular line overlying the sixth and seventh ribs was a mass 8 by 10 cm. which was tender, firm, indurated and only vaguely fluctuant. The skin over it was red, warm, and edematous. There was dullness over the entire chest posteriorly and flatness below the fifth rib posteriorly, and dullness below the third rib anteriorly. Over this area voice and breath sounds, and tactile fremitus were diminished to absent. The left border of cardiac dullness at the apex was in the anterior axillary line. A few coarse and numerous fine râles were heard above the area of flatness. There was an incomplete right inguinal hernia and an old scar from a laceration of the right thigh. The remainder of the physical examination was essentially negative.

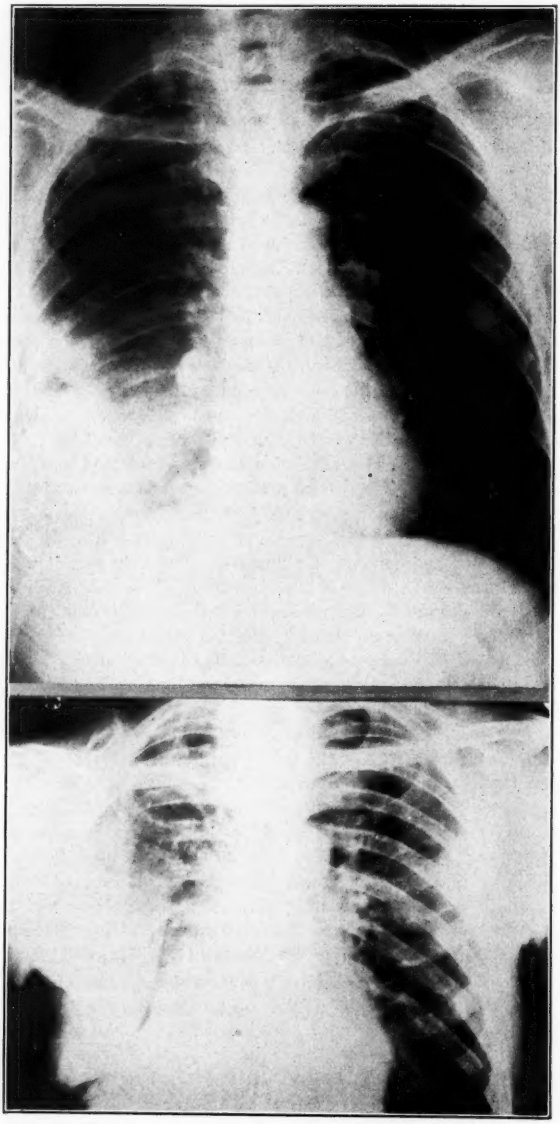
The blood pressure was 105/70, the urine contained a trace of albumin, and the blood showed hemoglobin 70 per cent, R.B.C. 3,800,000, and W.B.C. 18,000 with polymorphonuclears 68 per cent, monocytes 2 per cent, staph forms 5 per cent, lymphocytes 20 per cent, and eosinophils 5 per cent. The Wassermann and Kahn tests were negative. Repeated sputums were negative for tubercle bacilli.

Radiographic studies of the chest showed an area of increased density and obscuration of all lung markings laterally between the diaphragm and the fifth rib posteriorly. The bronchovascular markings were increased and there were several areas of increased density, probably Goan's tubercles. In the lateral films this area of density was definitely encapsulated and located in the posterior portion of the chest.

Roentgenograms made following aspiration (Fig. 1) showed an encapsulated collection of fluid with part of it, shown in the previous studies, replaced by air and a fluid level below which there was some undrained fluid.

A thoracentesis was done and 200 c.c. of thick red brown pus was obtained. Cultured on the ordinary media, no growth was obtained. On December 18, the indurated mass of the chest wall was incised and a small amount of sanguinopurulent fluid obtained which contained sulfur granules and from which actinomyces were cultured. Since the wound oozed and bled freely, it was packed with gauze and a pressure dressing applied. With nitrous oxide anesthesia two days later a segment of the seventh rib in the mid-scapular line was excised and the empyema

A.



B.

Fig. 1*A* and *B*.—Case 1. Roentgenograms taken before and after operation. Actinomycosis of the right chest wall and pleural cavity was drained by incision and thoracotomy, and later by Schede thoracoplasty. This was followed by electrosurgical cautery, and chemical destruction of infected tissue.

cavity drained. It contained approximately 400 c.c. of pus. From the pus were isolated sulfur granules, and from it actinomyces were cultured. At this time roentgen therapy and massive doses of iodine were started.

On Jan. 4, 1935, under nitrous oxide anesthesia a Schede thoracoplasty was done, removing segments of ribs five, six, seven, eight, nine, and ten, and uncovering completely a cavity partially covered with granulation tissue which bled freely. Since the patient was showing some evidence of shock the wound was packed with gauze and 500 c.c. of citrated blood was given.

Ten days later with nitrous oxide anesthesia the entire exposed surface was peeled with the cutting diathermy electrode, excising a wide layer from the chest wall and removing the pleura so as to expose the lung. Exposed rib stumps were also removed. In the angles at the junction of the visceral and parietal pleurae the tissue was destroyed with the actual cautery. The wound was packed with iodoform gauze.

Several cultures from the surface of the wound and from granulation tissue failed to grow actinomyces. It was believed, however, that successful eradication of the disease would be more likely by peeling the surface of its granulation tissue. Consequently, the procedure described above was repeated Feb. 26, 1935, approximately six weeks later. The cavity had become small. After controlling the bleeding points the entire surface was painted with 10 per cent formalin solution and the wound packed with wet gauze.

Five weeks later the patient was returned to his family physician for subsequent care. The wound gradually closed and for more than two years it has remained healed, and the patient has felt perfectly well. During this time he has gained forty-two pounds and has conducted most of his usual activities. Recent roentgenograms showed only the operative defect of the chest wall and some increased density presumably the result of a thickened pleura.

CASE 2.—P. C., a white male, farmer, aged 47, entered the University Hospital Oct. 2, 1935, complaining of fever, weakness, and pain in the left chest. He had been well until one month previously when he developed an upper respiratory infection with which there was a slight nasal discharge and a productive cough lasting a few days. A week later he developed constant pain in the left chest and on the following day he had a fever, 102°, weakness, malaise, and the pain became pleural in character. Pain and fever persisted though less severe, the appetite was poor with a weight loss of ten pounds, and the cough had been productive of a small amount of mucopurulent sputum which on two occasions was streaked with blood.

The family and past histories were negative. Questioning subsequent to the establishment of a diagnosis failed to elicit any animal or other source of contact with the disease. The patient was well developed and nourished, and appeared moderately ill. There was some anterior cervical adenopathy. The teeth were in good repair and there was no pyorrhea.

The left side of the chest lagged and moved much less than the right. Below the fifth rib posteriorly there was dullness to flatness and over the same area there was diminution of breath and voice sounds and of tactile fremitus. The heart was not displaced.

The blood pressure was 105/68. The abdomen, extremities, genitalia, and rectum were negative. On admission the temperature was 100.2, the pulse 110, and respirations 30. The urine was negative, and the blood showed hemoglobin 68 per cent, R.B.C. 4,300,000, W.B.C. 13,050, with polymorphonuclears 72 per cent, staph 2 per cent, lymphocytes 23 per cent, monocytes 2 per cent, eosinophils 1 per cent.

Radiographic studies showed the right lung and pleura to be clear. On the left side there was obscuration of the lower half of the left lung and of the diaphragm. The cardiac shadow was only slightly displaced to the right. Two attempts to aspirate fluid were unsuccessful.

Two weeks later some swelling with edema and redness of the skin had developed in the chest wall extending anteriorly from the posterior axillary line into the axilla and under the pectoral muscles. Roentgenograms showed an increase in the area of density, its upper border conforming to the interlobar fissure and its outer border extending along the chest wall between the sixth and tenth ribs in the posterior axillary line. The dome of the diaphragm on the left side appeared adherent to the lateral chest wall. There was also a shadow of soft tissue swelling of the left lateral chest wall.

On the following day the swelling of the chest wall and the pleural cavity were aspirated in the seventh interspace. Two hundred cubic centimeters of thick yellow

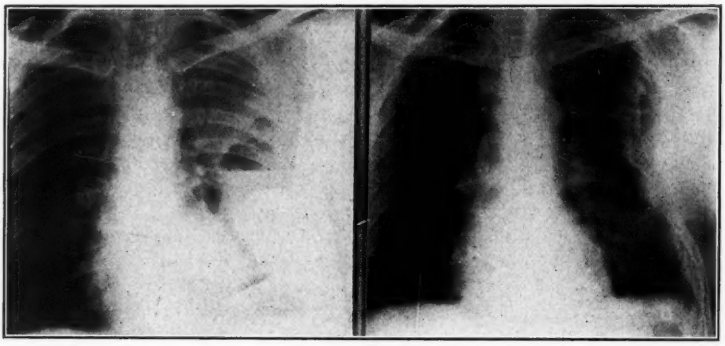


Fig. 2.—Case 2. Actinomycoses of the left pleural cavity and chest wall before and subsequent to surgical treatment. After widely incising the infected tissue of the chest wall and saucerizing the empyema cavity the infected tissue was excised as widely and completely as possible and the surface sterilized by electrothermal and chemical agents.

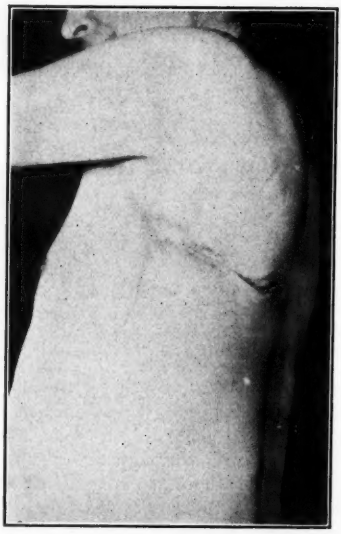


Fig. 3.—Case 2. Photograph of patient after complete healing had taken place. The wound has remained healed and the patient well for more than sixteen months.

and brown pus was obtained. The pus from both sites contained sulfur granules with mycelial filaments typical of actinomyces and on culture yielded the same organisms.

Iodine in quantities rapidly pyramiding to massive doses was given and an autogenous actinofiltrate was made and subsequently administered. The swelling and induration of the chest wall rapidly increased and extended, and the overlying skin became red and shiny.

Roentgenograms taken following aspiration (Fig. 2) showed an encapsulated pyopneumothorax measuring 6 by 9 cm. on the left side. The pleura over the superior surface appeared to be very thick, the surrounding lung tissue increased in density and the left leaf of the diaphragm adherent to the chest wall.

At this time the patient was seen in surgical consultation by me and transferred to the surgical service for subsequent treatment. On Nov. 5, 1935, under nitrous oxide anesthesia simple drainage was established. The chest wall was widely incised and the empyema cavity drained by costal resection. A moderate amount of thick reddish brown pus was evacuated from the chest wall and dark yellow pus from the empyema cavity.

On Dec. 2, 1935, a Schede thoracoplasty was performed resecting long segments of ribs three, four, five, six, and seven, uncovering the empyema cavity in its entirety. In the floor of the cavity there was a small excavated area which appeared to extend into the substance of the lung and communicated with a bronchus. The grayish tissue which lines this area was burned deeply with the actual cautery, and the thick visceral pleura which in areas was covered with granulation tissue was excised with a large loop electrode with the cutting current. The wound was then packed with gauze soaked with acriflavin. Following operation 600 c.c. of citrated blood was given.

Beginning a few days following operation a total of 400 R. units of x-ray was given to the exposed wound.

On Jan. 10, 1936, a month after the thoracoplasty, cultures of curettings of granulation tissue revealed the presence of actinomyces. The wound had diminished to approximately one-half its previous size and the patient's general condition had greatly improved.

On Jan. 12, 1936, under nitrous oxide anesthesia, a layer of tissue was removed from the entire exposed surface with an electro cutting loop. The underlying tissues which in many areas were normal in appearance were seared by electro-coagulation. The operation was followed by a second transfusion of 500 c.c. Although all subsequent cultures failed to grow actinomyces, further effort to rid the wound of this organism was made. Consequently on Jan. 18, 1936, the operative procedure as described above was repeated.

The patient was dismissed from the hospital four weeks later and returned in six weeks with the wound entirely closed. It has remained healed and he has felt perfectly well to date, sixteen months following final closure of the wound. He has gained thirty pounds and for nearly a year has worked daily.

STAB WOUND OF THE HEART

Report of a Case Treated Conservatively*

John W. Strieder, M.D.
Boston, Mass.

IN VIEW of the remarks by Dr. Dan Elkin in the discussion of the recent paper by Bigger,[1] the following case of stab wound of the heart, with recovery without operative intervention, seems worthy of report.

A 12-year-old Armenian schoolboy, said to have been stabbed accidentally by a schoolmate with a "switch-blade" knife, was admitted to the Second Surgical Service, Boston City Hospital, at 4 P.M. April 12, 1938. He was bleeding slightly from a ½ inch incised wound in the fourth left intercostal space, one inch to the left of the sternum. Although conscious and rational, he presented the classical picture of cardiac tamponade. The heart sounds were muffled and distant. The pulse rate was 120, and the blood pressure 48/20.

There was dyspnea and cyanosis, but the neck veins were not visibly engorged.

The area of cardiac dullness was not enlarged to percussion, but a portable roentgenogram of the chest showed a marked increase in size of the heart shadow typical of pericardial enlargement by fluid.

Immediate operative interference was obviously indicated. While the operating room was being set up, having in mind Dr. Bigger's suggestion, a needle was introduced into the pericardium in an attempt to reduce the tamponade and also to take the pressures.

The 16 gauge needle was inserted through the fifth left interspace just outside the midclavicular line and 10 c.c. of blood withdrawn from the pericardium. An Ayer spinal fluid manometer, with the system full of sterile normal saline solution, was attached to the needle. The column reached equilibrium at 135 mm. on several trials.

One hundred cubic centimeters of blood was then withdrawn and introduced into the patient's median basilic vein. There was immediate and dramatic improvement in his condition. Within a few minutes the blood pressure rose to 140/60. The dyspnea and cyanosis disappeared, and the patient's general appearance was greatly improved.

Since there was no evidence of a recurrence of the tamponade during the next hour, it was considered safe to observe the patient closely, maintaining the operating room in readiness.

The patient exhibited no further signs of tamponade or concealed hemorrhage.

The next morning the lower half of the chest was dull and 100 c.c. of blood was removed from the left pleural cavity through the eighth interspace in the posterior axillary line. This, also, was placed in the vein. No blood could be obtained from the pericardium at this time. There was no evidence of pneumothorax in the portable roentgenogram at this time.

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*From the Second Surgical Service, The Boston City Hospital.
Received for publication, December 5, 1938.

The electrocardiogram April 18 showed slight changes which were believed to be consistent with acute myocardial injury. From this date until May 17, the electrocardiogram went through a series of changes characteristic of an acute myocardial injury (such as coronary thrombosis). On May 17 there were striking changes toward normal.

Clinically the patient's temperature reached normal on April 16, and he maintained a normal chart from this time onward.

The wound healed without sepsis, and the patient was discharged from the hospital May 26, 1938.

He was last seen Oct. 15, 1938. At this time electrocardiogram and physical examination were normal, and he had no symptoms. He had been at school since the beginning of the term in September.

COMMENT

It is not believed that this patient had an actual wound of the myocardium which entered the cavity of the heart. It seems more likely that a coronary artery or vein was wounded and the myocardium was incised for only a portion of its thickness.

Although this patient recovered without operation, it is believed that the dangers of future aneurysm of the heart or of a coronary artery are real. Elkin* believes that undoubtedly some patients will recover under conservative methods, and, if they are in good condition and the tamponade not progressive, aspiration may be done. However, it would seem that not operating upon a patient with a stab wound of the heart is a very hazardous course to follow.

Thanks are due to Dr. James M. Faulkner for his interpretation of the many electrocardiograms in this case.

REFERENCE

1. Bigger, I. A.: Heart Wounds, J. THORACIC SURG. 8: 239, 1939.

*Personal communication.

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The American Association for Thoracic Surgery

Twenty-Second Annual Meeting

Los Angeles, Calif., July 5, 6, and 7, 1939

Program

WEDNESDAY, JULY 5

9:00 A.M. Business session.

9:30 A.M. Scientific session

1. Benign Chondroma of the Chest Wall.
   Fred R. Harper, Denver, Colo.
2. Primary Tumors of the Ribs.
   Robert M. Janes, Toronto, Can.
3. Pectus Excavatum.
   A. Lincoln Brown, San Francisco, Calif. (by invitation)
4. Primary Tumors of the Diaphragm.
   Joseph W. Gale and (by invitation)
   Stanley R. Edwards, Madison, Wis.
5. Removal of Teratoma from Anterior Mediastinum.
   (Moving picture demonstration.)
   Stuart W. Harrington, Rochester, Minn.

2:00 P.M. 6. Address: Pulmonary Ventilation and Anesthesia in Major Chest Surgery.
   Dr. Clarence Crafoord, Stockholm, Sweden.

7. Differential Pressure and Reduced Lung Function in Intrathoracic Operations.
   W. E. Adams, Chicago, Ill.
8. Further Studies on Survival Following the Maintenance of Life During Experimental Occlusion of the Pulmonary Artery.
   John H. Gibbon, Jr., Philadelphia, Pa.

9. Hemicardiac Hypertrophy and Enlargement Solely Due to Increased Peripheral Resistance: A Study of Pulmonic and Aortic Stenosis Experimentally Produced.
   Emile Holman, San Francisco, Calif.
10. Factors Affecting the Regeneration of the Lung.
    Roy Cohn, San Francisco, Calif. (by invitation).

THURSDAY, JULY 6

9:00 A.M. Scientific session

11. A Selective Type of Thoracoplastic Operation.
    William A. Hudson, Detroit, Mich.
12. The End Results in Approximately 500 Thoracoplasty Patients.
    E. J. O'Brien, Detroit, Mich.
13. Extrafascial Air as an Adjunct to Thoracoplasty with Extrafascial Apicolysis.
    T. B. Aycock and (by invitation)
    Otto C. Brantigan and Hugh Welch, Baltimore, Md.
14. Planography (Body Section Radiography) as Applied Especially to Pulmonary Disease.
    Warren C. Breidenbach, Dayton, Ohio (by invitation).
15. Four Years' Experience with Extrapleural Pneumo-Oleothorax.
    Oscar S. Proctor, Seattle, Wash.

1:00 P.M. Executive session.

2:00 P.M. Presidential Address. Two Interesting Benign Lung Tumors of Contradictory Histopathology. Remarks on the Necessity for Maintaining the Lung Tumor Registry.
Harold Brunn, San Francisco, Calif.

FRIDAY, JULY 7

9:00 A.M. Scientific session

16. A Comparative Report on Infection of Thoracoplasty Wounds: Experiences with the Ultraviolet Irradiation of Operating Room Air.
    Richard H. Overholt and (by invitation)
    Reeve H. Betts, Boston, Mass.
17. Polypoid Bronchial Tumors.
    H. Brodie Stephens and (by invitation)
    Alfred Goldman, San Francisco, Calif.
18. The Diagnosis and Surgical Therapy of Patent Ductus Arteriosus.
    John C. Jones, F. S. Dolley, and (by invitation)
    Lewis T. Bullock, Los Angeles, Calif.
19. Operation for Development of Collateral Circulation to the Heart.
    Peter Heinbecker and (by invitation)
    Wesley A. Barton, St. Louis, Mo.
20. Dorsal Sympathetic Ganglionectomy for Intractable Asthma.
    Duane Carr, Memphis, Tenn.

2:00 P.M. Scientific session

21. Large Infected Pulmonary Cysts Simulating Empyema.
    Herman C. Maier, New York, N. Y.
    Cameron Haight, Ann Arbor, Mich.
22. Acute Putrid Abscess of the Lung.
    Harold Neuhof and Arthur Touroff, New York, N. Y.
23. Joint Manifestations Associated with Lung Tumors.
    Willard Van Hazel, Chicago, Ill.

PAPERS TO BE READ BY TITLE

1. Studies of the Jugular, Carotid, and Pulmonary Pressures of Anesthetized Dogs During Positive Inflation of the Lungs.

George H. Humphries (by invitation), Richard L. Moore, and Howard Barkley (by invitation), New York, N. Y.

2. Chronic Inflammatory Lesions of the Lung Simulating Primary Bronchogenic Carcinoma.

S. O. Freedlander and (by invitation), S. E. Wolpaw, Cleveland, Ohio.

3. Diffuse Polypoid Laryngotracheobronchitis with a Preliminary Note on Endoscopic Curettage.

Paul C. Samson, Oakland, Calif.

4. Hemothorax: Its Relation to Carcinoma of the Lung.

Henry L. Cabitt, Boston, Mass.

5. Carcinoma of the Lung with Associated Tuberculosis.

Thomas J. Kinsella, Minneapolis, Minn.

6. Pleurolysis with Flexi-Tissue and Vioform Pack for Closure of Residual Cavities Remaining after Thoracoplasty.

Ralph C. Matson, Portland, Ore.